

# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—28TH YEAR.

SYDNEY, SATURDAY, MAY 3, 1941.

No. 18.

## Table of Contents.

(The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.)

ORIGINAL ARTICLES—	Page.	MEDICAL SOCIETIES—	Page.
Modern Aspects of Puerperal Sepsis, by Arthur M. Hill	537	Melbourne Paediatric Society	559
Report on Cerebro-Spinal Meningitis, by Lieutenant-Colonel M. J. Holmes, D.S.O.	541	EAR PICKING AND EYE CLEANING IN THE MIDDLE AND FAR EAST	562
Meningococcal Infection with Special Reference to Meningococcal Septicæmia, by Gerald C. Moss, M.B., M.R.C.P., F.R.A.C.P.	548	THE TOURNIQUET AND BOTH BONES	563
NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—		THE ILLAWARRA SUBURBS MEDICAL PRACTITIONERS' NATIONAL EMERGENCY SERVICE	563
Regional Anatomy	552	NAVAL, MILITARY AND AIR FORCE—	
LEADING ARTICLES—		Appointments	563
Australian Doctors and the War	553	AUSTRALIAN MEDICAL BOARD PROCEEDINGS—	
CURRENT COMMENT—		New South Wales	563
Gout, the Forgotten Disease	554	OBITUARY—	
The Iodine Content of the Blood	555	John Ferguson Chambers	564
ABSTRACTS FROM MEDICAL LITERATURE—		Zelman Schwartz	564
Radiology	556	MEDICAL APPOINTMENTS	564
Physical Therapy	557	BOOKS RECEIVED	564
BRITISH MEDICAL ASSOCIATION NEWS—		DIARY FOR THE MONTH	564
Scientific	558	MEDICAL APPOINTMENTS: IMPORTANT NOTICE	564
EDITORIAL NOTICES			564

### MODERN ASPECTS OF PUERPERAL SEPSIS.<sup>1</sup>

By ARTHUR M. HILL,

Honorary Obstetric Surgeon, Women's Hospital,  
Melbourne.

RECENT years have seen two valuable additions to our knowledge of puerperal sepsis. The first is a more exact appreciation of its bacteriology and morbid anatomy. The second is the development of a new and potent form of chemotherapy.

#### Conception of Puerperal Sepsis.

Today we regard puerperal sepsis as a wound infection. In the uterus of every parturient woman there is an open wound. This wound, the placental site, larger in area than the palm of a man's hand, is honeycombed with sinuses containing blood clot, an ideal pabulum for bacteria. During labour a woman may sustain further wounds of the cervix, vagina, vulva or perineum. If, during labour or the early puerperium, pathogenic organisms gain entrance to such wounds, the patient faces the dangers of puerperal sepsis.

#### Bacteriology.

The organisms responsible for the majority of severe and fatal puerperal infections are the haemolytic streptococcus, the anaerobic streptococcus, *Staphylococcus aureus* and *Clostridium welchii*.

Less important are *Bacillus coli*, the non-haemolytic streptococci, *Streptococcus viridans*, *Staphylococcus albus*

and the gonococcus, the first two of which are fairly commonly recovered from the vagina in low-grade sepsis, not infrequently in association.

Let us consider the important microorganisms in more detail.

#### *Hemolytic Streptococci.*

A large proportion of the known strains of haemolytic streptococci can be placed in one or other of a number of serological groups designated A, B, C, D, E, F, G, H and K. Organisms of the groups A, B, C and G are capable of producing human puerperal infection, those of group A easily predominating as a cause of both mild and severe sepsis.

In a series of 90 consecutive cases of puerperal infection with haemolytic streptococci investigated at the Women's Hospital, in 66 the infecting strain was group A, in nine it was group B, in nine it was group C and in six it was group G.

The great importance of group A haemolytic streptococci lies not only in their widespread incidence, but in their infectivity, and in the invasive power of many of them. For the production of puerperal sepsis it is usually only necessary to introduce these organisms into the genital tract of the mother. The virulence of the individual organism then determines clinical infection.

What is the source of group A haemolytic streptococci? They are so rarely present in the vagina at or near term that infection from the mother's own genital tract can be almost eliminated as a likelihood. They cannot be recovered from the perineal skin or faeces of healthy women. They can, however, be recovered from the upper respiratory tract of between 5% and 10% of the general population, the proportion being higher in winter time, and in the subjects of recent colds, sore throats, antrum infections or "influenza". They have been isolated from the hands

<sup>1</sup> Read at a meeting of the Victorian Branch of the British Medical Association on March 5, 1941, at Melbourne.

of nearly 4% of normal women. They have been found to survive in the dust of an infected room for as long as ten weeks. Finally, they are well known as a cause of pharyngitis, tonsillitis, erysipelas, scarlet fever, *otitis media*, impetigo and other skin infections. This knowledge of their power and distribution is essential to the exercise of an intelligent prophylaxis.

Group B streptococci are frequently inhabitants of the normal vagina and throat.

The source of infection with streptococci of groups C and G is most likely the human throat.

#### *Anaerobic Streptococci.*

Anaerobic streptococci are normal inhabitants of the vagina in the last months of pregnancy in between 15% and 40% of women. Our experience at the Women's Hospital points to the lower figure; but in the early months of pregnancy the incidence is higher.

Invasion of the body by the anaerobic streptococcus would appear to depend less on the virulence of the individual organism than on lowering of local or general resistance, or of both, in the mother. Here, at once, is a big difference from the haemolytic streptococcus.

Damage is one key to invasion, and in our experience extensive bruising and injury to the tissues are not more important than general maternal damage in the form of blood loss. Severe anaemia, most often the result of haemorrhage, has been too frequent a feature of our grave cases to be overlooked.

#### *Staphylococcus aureus.*

The common source of *Staphylococcus aureus* is the skin, whether of the mother, of her associates or of her medical attendants. Particularly dangerous are boils and carbuncles, which may readily initiate a severe or fatal infection. *Staphylococcus aureus* may be present in the skin in the absence of a demonstrable lesion, which explains its incidence as a cause of breast abscess and of wound infection following Cæsarean section.

#### *Clostridium Welchii.*

*Clostridium welchii* is of a widespread distribution, being recoverable from soil, dust, the sanitary pads sold as "sterile" by many firms, and the large intestines of most people. This last is the usual source of human puerperal infection.

*Clostridium welchii* shares with the anaerobic streptococcus the necessity of maternal damage before it can invade the body. While general fatigue, toxæmia and blood loss are an advantage to it, local tissue damage, and in particular tissue death, pave the way for its invasion.

For that reason one anticipates the possibility of infection with *Clostridium welchii* in such conditions as prolonged or difficult labour, "failed forceps" delivery, premature rupture of the membranes, tubal induction of labour, plugging of the vagina, and any considerable mechanical interference, particularly when associated with a dead fetus *in utero*.

#### *Bacillus coli.*

*Bacillus coli* is a normal inhabitant of the bowel, and its infection of the genital tract most often occurs after mechanical interference. In puerperal sepsis it is fairly commonly associated with other organisms, notably the anaerobic streptococcus and the non-haemolytic streptococcus; but of itself it is not often a cause of severe infection. Its frequency as a cause of puerperal pyelitis, however, is well known.

#### *Morbid Anatomy.*

The method of spread and the reactions in the body vary so greatly with the different microorganisms that it is necessary to consider the morbid anatomy of puerperal infection separately for each bacterial type.

The following description deals with salient characteristics.

#### *Infections with Streptococcus Haemolyticus Group A.*

In the presence of infection with a group A haemolytic streptococcus the lesions in the birth canal tend to show relatively little inflammatory reaction. Infected cervical and vaginal lacerations appear indolent and are covered with a layer of exudate containing streptococci. The uterus is generally well involuted, and the decidua appears smooth and clean. Microscopic examination may reveal streptococci between the muscle bundles, often with inadequate leucocytic response.

Spread beyond the uterus, which occurs with highly virulent strains, is by anatomical continuity irrespective of tissue boundaries, so that the pelvic portion of the peritoneum and cellular tissue are usually involved together.

Infection of the cellular tissue, as also of the Fallopian tubes and ovaries, is accompanied by oedema. In the Fallopian tubes the fimbriated ends are not occluded nor is their lumen distended, so that pyosalpinx is rare.

The peritoneum frequently fails to localize the infection, and in fatal cases the abdomen may contain pints of thin, turbid fluid, and there may be few or no adhesions between loops of bowel. In the most fulminant cases even lymph flakes may be absent, and the peritoneal lustre of the bowel may be unimpaired.

Spread of infection to the blood stream is common with virulent strains, and is due to the breaking down of infected thrombi in the small veins of the uterine wall. With a continuance of this process showers of streptococci pass into the general circulation, producing the clinical picture of a septicæmia in which pyrexia and tachycardia are more or less sustained.

In most cases in which the patient survives for more than ten days small abscesses up to four or five millimetres in diameter are found in the uterine wall, most often beneath the placental site.

Owing to the fibrinolytic action of the streptococci in liquefying blood clot, thrombus formation in large vessels, such as the ovarian and iliac veins, is not common. For the same reason embolic lesions are rare.

#### *Infections with the Anaerobic Streptococci.*

In infections with anaerobic streptococci the cervix, vagina or perineum may be lacerated and bruised. The wound surfaces are acutely inflamed and the edges may be necrotic, while there may be a foul-smelling discharge. The uterus is generally subinvolved and contains offensive disintegrating blood clot. The decidua is thickened and shaggy, and appears "unclean".

With further invasion abscesses may form in the uterine wall, these usually being larger than those seen in haemolytic streptococcal infections; rarely such abscesses may cause the peritoneal coat to bulge.

Owing to the slow rate of spread and the pronounced tissue reaction, involvement of the cellular tissue produces a brawny hard induration; and peritonitis is usually pelvic, while well-marked adhesions are present between adnexa and coils of gut. General peritonitis is rare, but may result from the rupture of an abscess in the uterine wall or broad ligament.

In serious infections the characteristic feature is a spreading thrombophlebitis, which involves the iliac veins and *vena cava* more commonly than the ovarian veins. Failing organization and resolution, pieces of friable, infective blood clot break off into the general circulation, with striking clinical effects. The septic emboli are arrested in the lungs, and in fatal cases it is usual to find the lung fields sparsely studded with pulmonary abscesses varying in size from that of a pea to that of a cherry, each with its fibrous tissue capsule and central area of necrosis. This process of septic embolism causes shock and a typically intermittent fever, and if prolonged over some weeks is likely to prove fatal.

#### *Infections with Staphylococcus aureus.*

Although it is most often responsible for mild and localized infections, *Staphylococcus aureus* may cause a swiftly fatal septicæmia or general peritonitis.

Lesions in the genital tract produce an acute inflammatory reaction, with purulent discharge. Invasion of the uterine wall typically produces a number of minute abscesses, and this same tendency is seen in other organs should invasion of the blood stream occur. Thus in a fatal case of septicæmia, miliary abscesses may be present in the heart muscle, kidneys and liver. Bacterial embolism may cause petechial spots in the skin. In less acute blood-borne infections, metastatic abscesses are apt to develop in the subcutaneous tissues, skeletal muscles and joint cavities.

The role of *Staphylococcus aureus* in producing mastitis and wound infection after Cæsarean section has already been mentioned.

#### Infections with *Bacillus Coli*.

Lacerations infected with *Bacillus coli* produce a pronounced inflammatory reaction, with offensive purulent discharge. When, as may occur after considerable tissue damage, infection spreads beyond the uterus, it is as a rule confined to the cellular tissue and peritoneum, which react strongly, and there is a tendency to subsequent abscess formation.

Isolated cases have been recorded of localized gangrene of the uterine wall; but such virulence is exceptional.

Invasion of the blood stream is not particularly uncommon, but fatal septicæmia is rare. Of fatalities recorded in the past, many without doubt have been due to accompanying invaders, notably anaerobic streptococci.

#### Infections with *Clostridium Welchii*.

Bruising and laceration of the cervix, vagina or perineum are generally present in infections with *Clostridium welchii*. The wound surfaces are grey and necrotic, and there is a brown musty discharge; in a mixed infection the tissues around the wound are acutely inflamed and the discharge is foul.

Conditions in the uterus depend on whether or not delivery has occurred. During labour the fetus is commonly dead, or even macerated, and its tissues may be distended with gas. After delivery the uterus is large and atonic and its contents are at varying stages of a liquefying necrosis. The decidua is softened, shaggy and filthy looking. Placental tissue, if present, is a dirty grey, parts of it are becoming paste-like in consistency, and it exudes a slightly pungent cadaveric odour. The lochia is brown and profuse and smells cadaveric or foul.

Invasion of the uterine muscle, or physometra, produces a swiftly spreading cell death, and aggregations of the bacilli may lie between the muscle bundles with a minimum of leucocytic response. At death or operation the atonic uterus is lustreless and of a mottled grey or bluish grey colour; its large veins often bulge with black thrombi, and fine bubbles of gas may be expressed from the cut muscle surface. In cases associated with blood destruction the uterus shares with other tissues something of the jaundice, which in the skin may produce any shade between muddy yellow and bronze.

Peritonitis, which almost invariably accompanies physometra, is typically pelvic, and is associated with a thin brown exudate laden with organisms.

After invasion of the uterine muscle death is the rule; and the overwhelming destructive power of *Clostridium welchii* toxin is seen in an extensive cell degeneration through the liver, kidneys, myocardium and spleen. Numbers of bacilli may be widely scattered through the parenchyma of these organs, usually with little evidence of leucocytic response. In the kidneys there is a hyperacute nephrosis, chiefly involving the tubules, with the added factor, in cases associated with gross haemolysis, of a widespread deposition of blood pigments in the tubules.

Except in the uterus removed at operation, gas is rarely detected in the tissues during life; but within a few hours of death the liver and spleen may be honeycombed with gas, and gas bubbles may be present in the myocardium and heart's blood, and less often in the skeletal muscles and subcutaneous tissues.

#### Investigation.

At the onset of fever in the puerperium the patient's history is reviewed and she is subjected to a gentle but thorough general examination. In this way upper respiratory and pulmonary infections, alimentary lesions and general infectious diseases can usually be quickly detected or eliminated. Clinical evidences of mastitis and pyelitis are then sought, and even in the absence of pyelitic signs a catheter specimen of urine is withdrawn for microscopic and bacteriological examination. Attempted culture from the urine is of additional value if infection with *Clostridium welchii* is suspected.

The examination concludes with the incubation on nutrient media, both aerobic and anaerobic, of material from the vagina, and, in addition, with aerobic and anaerobic incubation of specimens of blood in any case in which the temperature reaches 101° F. or the pulse rate is unduly quickened.

Vaginal swabblings are taken without the patient's being moved; the labia are simply separated and the swab is passed well up the posterior vaginal wall.

Digital vaginal examination is not carried out as a routine measure in the first week of a puerperal infection, because there is then little of value it can reveal, and it carries the dual dangers of transferring further infection to the upper part of the genital tract and of disturbing the body's attempts at localizing the infection.

Inspection will reveal infection of the vulva, vagina or perineum and the necessity for releasing sutures.

#### Clinical Types of Infection.

When the presence and bacterial nature of puerperal infection have been diagnosed it is necessary to determine its anatomical site and extent. Thus there are a number of clinical types or grades of puerperal sepsis, of which the chief are the following:

*Infection Localized to the Birth Canal.*—Infection localized to the birth canal is the simplest type, and fortunately by far the commonest. The most frequent lesion is septic endometritis. The patient does not usually feel ill, and except for perhaps an isolated exacerbation, the pyrexia and increase in pulse rate are of a low order. Almost all these patients are afebrile within a week.

The correct bacteriological diagnosis bears no reliable relationship to such signs as uterine subinvolution or offensive lochia. As all these infections are due to pathogenic organisms, it is time the old unscientific term "sapromyia" was abandoned.

*Infection Spreading Beyond the Uterus but Confined to the Pelvis.*—Infection spreading beyond the uterus but confined to the pelvis includes pelvic peritonitis (with salpingo-oophoritis), pelvic cellulitis and thrombophlebitis. Spread to the peritoneum and cellular tissue is suspected when, usually between the third and tenth days of infection, there is an increase in the pyrexia and in the pulse rate, and at the same time fairly acute lower abdominal pain occurs, accompanied by distension, tenderness and muscle resistance. Within a few days an ill-defined mass may be palpable in one or both iliac fossæ, and it may be possible to distinguish peritonitis from cellulitis. In peritonitis the mass tends to be more discrete, ovoid and high in the pelvis, and pain is usually more prominent. In cellulitis the mass is less clearly defined, at first is only low in the pelvis, and is felt as an induration spreading fanwise out to the lateral pelvic wall. A finger in the rectum may disclose utero-sacral infiltration. Thrombophlebitis is to be suspected when, in the absence of signs of extrauterine extension, fever continues for longer than ten days, and more particularly so if the infecting organism is an anaerobic streptococcus. *Phlegmasia alba dolens* is an example of slow spread to the femoral vein of a mild pelvic thrombophlebitis.

*General Peritonitis.*—General peritonitis, a grave complication, is apt to occur early. It is to be suspected when the general condition of a patient with a raised temperature and pulse rate fairly rapidly deteriorates; the pulse rate quickens, the tongue becomes drier, the temperature perhaps falls after a rigor, and at the same time there is increasing abdominal distension. The attack may be

ushered in with brisk diarrhoea. Deep abdominal tenderness, particularly "release tenderness", is usual; but abdominal rigidity is absent and spontaneous pain a rarity.

The combination of increasing abdominal distension, with deterioration in the patient's general condition, is characteristic and usually the first indication. Later come rapid respirations, muddy facies, vomiting and peripheral circulatory failure.

**Septicæmia.**—Septicæmia is suspected in the presence of high fever and a rapid pulse rate, particularly when these signs are sustained and are not adequately explained by other lesions.

Certain organisms, as already indicated, tend to produce distinctive clinical pictures. Thus after normal labour without even a vaginal examination, haemolytic streptococci may cause remittent or continuous high fever, persistent tachycardia and a severe or rapidly fatal illness. Anaerobic streptococci may produce after complicated labour an intermittent pyrexia and tachycardia, with multiple rigors and a more prolonged illness. *Clostridium welchii* may produce after complicated labour the excruciating pain and collapse of phynometra, or a deepening jaundice or, with or without either antecedent, a rapidly increasing pulse rate, pallor, a minimum of fever and early failure of the peripheral circulation.

But "characteristic" pictures are by no means the rule, and more often than not one cannot judge with accuracy from clinical signs the nature of the infecting organisms. The diagnosis of septicæmia depends ultimately, and very often solely, upon the results of attempted blood cultures. Aerobic and anaerobic incubation of blood in nutrient media should therefore be made at the earliest suspicion of invasion of the blood stream. Particularly in the case of anaerobic streptococci it may be necessary for the attempts at cultivation from the blood to be repeated daily for several days to obtain a positive result.

#### Preventive Measures.

Preventive measures begin with the institution of efficient antenatal care.

Here mention will be made only of the importance of combating maternal malnutrition and anaemia, mental and bodily fatigue in the last weeks, respiratory and intercurrent infections, and skin lesions, such as impetigo, paronychia and furunculosis. The elimination of dental sepsis is of special importance in cardiac cases. The mother must be made aware of the dangers of infective processes in herself and her family, and taught to report their occurrence.

#### The Wearing of Masks.

Efficient face masks should be worn by all present at the confinement, and by the attendant at every exposure of the vagina until the end of the fifth day of the puerperium. The mother who has a respiratory infection during labour should also be masked. Masking must never be used as an excuse to keep on duty a person whose throat is known to be infected. An efficient mask covers both the nose and the mouth and passes well under the chin; it is so shaped as to prevent any side-flow of droplets, and is of a sufficient number of layers of a suitable material to arrest all types of droplets that can impinge on it from the respiratory tract. The Jessop hospital mask, which is used at the Women's Hospital, is efficient on all these counts.

#### Antiseptics.

"Dettol" is probably the most efficient antiseptic, both for gloved hands and for the patient's skin. It should be used in strengths of not less than 30%; "Dettol Cream" is excellent for the purpose. Weaker solutions, as Colebrook has pointed out, do not give the prolonged antiseptic effect which is so desirable.

During labour it is a wise precaution to smear with "Dettol Cream" every four hours the hands of any woman who has a respiratory infection, or of one under the influence of a disorientating narcotic (such as scopolamine and the barbiturates).

#### Conduct of Labour.

During labour the avoidance of trauma, blood loss and unnecessary fatigue must be a constant aim, and is the first line of defence against anaerobic infections.

Vaginal examinations are still too frequently carried out in normal cases. They are indeed rarely necessary in uncomplicated labour, for the information required can generally be obtained by abdominal and post-anal examination. The danger of prolapse of the cord associated with rupture of the membranes does not exist if the fetal head is fixed at the time of their rupture.

Sit-down baths must not be given during labour to *multiparae* or to any woman whose membranes have ruptured.

#### Antiserum.

*Clostridium welchii* antitoxin, in doses of 20,000 to 40,000 international units, is given as a routine measure at the Women's Hospital in cases of tubal induction of labour, plugging of the vagina, "failed forceps" delivery and dead *fetus in utero* if associated with interference or a foetid discharge. Incubation of material from the vagina may prove of great value in indicating future danger.

#### Sulphanilamide.

The prophylactic value of sulphanilamide in such cases as those mentioned above is worthy of trial, and sulphanilamide should certainly be employed when there has been exposure to the known risks of haemolytic streptococcal infection.

#### Prevention of Spread.

As soon as the diagnosis is made the patient suffering from puerperal sepsis should be isolated. When the infecting organism is a haemolytic streptococcus or *Staphylococcus aureus*, her previous room should be thoroughly disinfected and material should be taken from the upper respiratory tract and skin lesions of all contacts for incubation in nutrient media.

#### Personal Factor.

Finally, as Colebrook<sup>10</sup> has stated, the personal factor is perhaps the most important factor of all. In his words, "we shall not get rid of these occasional disasters unless we can count upon a high standard of intelligent vigilance and conscientious work". Our best defence is still an enlightened attention to detail.

#### Treatment.

The present-day treatment of puerperal sepsis can be considered under three heads: general treatment, chemotherapy and the treatment of individual lesions.

#### General Treatment.

The importance of good nursing should not require emphasis. The patient's room should be well aired and preferably open to sunlight. The diet should be liberal and nutritious, and the addition of vitamin A is probably of value. One should aim at a fluid intake of five pints daily, and patients with pronounced toxæmia, repeated vomiting or dehydration may require the intravenous administration of glucose in saline solution.

The patient is nursed in Fowler's position to aid uterine drainage. The bowels may be assisted with bland aperients, such as liquid paraffin. Purging is to be avoided, for it is weakening, may seriously interfere with local defences, and predisposes to sulphæmoglobinæmia a patient receiving sulphanilamide. Sufficient rest and sleep are of the utmost importance, and if they are not procurable with such drugs as chloral and the barbiturates, opiates should be used.

Anæmia is very common; yet it is necessary to emphasize that it is the clinical sign which is most often overlooked or insufficiently appreciated. It should be treated with massive doses of iron, preferably of the ferrous salt, aided, when the haemoglobin level has fallen to 50%, by blood transfusion, which should be repeated as indicated.

#### Chemotherapy.

The discovery of the sulphanilamide group of drugs has been the greatest therapeutic advance of this generation. Sulphanilamide consists of a benzene ring, to

opposite poles of which are attached an amino group ( $\text{NH}_2$ ) and a sulphonamide group ( $\text{SO}_2\text{NH}_2$ ). By the introduction of substituents into the amino group are derived such compounds as "Prontosil Red", "Prontosil Soluble", "Rubiazol", "Proseptasine" and "Suluseptasine". By the introduction of substituents into the amide group are produced "Uleron", "Albucid" and sulphapyridine ("M & B 693"). Sulphanilamide apparently acts by the selective inhibition of a metabolite (according to Fildes<sup>10</sup> and his co-workers, *p*-amino benzoic acid), which is essential to bacterial growth.

After oral administration sulphanilamide is rapidly absorbed from the small intestine and reaches its maximal concentration in the blood in three or four hours. The concentration then falls gradually to zero over the next twenty-four hours, excretion being by the kidneys.

To obtain the best therapeutic effect it is necessary to maintain a constant concentration of the drug in the blood. It should therefore be given at intervals of not more than six hours. In severe infections a blood concentration of between 8 and 14 milligrammes per 100 cubic centimetres is required, 10 milligrammes or more usually being optimal. To attain this last figure the dose is one gramme per day to 20 pounds of body weight—that is, seven grammes (14 tablets) for a woman weighing ten stone. This dosage is maintained for two or three days and then reduced by one or two grammes, so that at the end of a week the patient is receiving four to six grammes per day. Administration of the drug must be continued until after the temperature has returned to normal and until no organisms can be cultured from the blood, unless toxic effects demand its withdrawal. No patient should be allowed to continue receiving the drug for more than ten days without an estimation of the numbers of red and white cells, and thereafter these estimations should be repeated every third day. In the presence of haematuria and oliguria the dosage should be reduced. In the presence of a rash, secondary pyrexia or reduction in the numbers of white or red cells, the drug must be withdrawn. If it has not produced therapeutic effects within ten days it is not likely to prove efficacious and it should be discontinued. Later one may recommence the course with a different member of the series.

While sulphanilamide is being given, amidopyrine, sulphates and sulphur-containing foods should be withheld and purging must be avoided. So long as a patient retains the drug taken orally, it is quite unnecessary to give soluble forms by injection.

The most valuable compound in puerperal sepsis is sulphapyridine ("M & B 693"), which is potent and has a wide range. Pending recovery of the causative organisms by cultural methods, it should be given in any case of puerperal sepsis which threatens to be of severe grade. If haemolytic streptococci, *Clostridium welchii* or *Bacillus coli* are recovered, the giving of the drug is continued. Delay in administration, too small a dosage and too early withdrawal are still the common errors in its employment. The drug is probably best given crushed in milk, with the addition of 10 grains of sodium bicarbonate to each dose.

In haemolytic streptococcal and *Bacillus coli* infections "Prontosil" and sulphanilamide at times give equally good results.

In infections due to *Staphylococcus aureus*, sulphathiazol is of greater value than sulphapyridine. Recently, however, Osgood, Joski and Brownlee,<sup>11</sup> in the United States of America, have made a strong case for the use of inorganic arsenic, accompanied for the first few days by sulphathiazol. The dosage of arsenic is 1 part in 150,000 of the body weight for the first day; that is, for a patient weighing 60 kilograms, 0.4 gramme of "Neoarsphenamine" is given in divided doses. Three-quarters of this dose are given each subsequent day, and regular estimations of the blood arsenic content are made as a guide. In severe cases the blood concentration of arsenic aimed at is between 1 in 150,000 and 1 in 200,000.

There is no chemotherapy of known value for anaerobic streptococcal infections.

#### Treatment of Individual Lesions.

Time will permit only the mention of certain points of importance in the treatment of individual lesions.

**Septic Endometritis.**—The principles of treatment in septic endometritis are rest and drainage. Ecblolic drugs, and rarely the intrauterine instillation of glycerine, may be indicated for the subinvolved uterus with retained, offensive or purulent discharge, and associated most often with anaerobic or coliform infection. Ecblolic drugs and the intrauterine instillation of glycerine should not be used, however, in haemolytic streptococcal infections.

**Infections Localized to the Pelvis.**—Two valuable measures in infections localized to the pelvis are the application of heat and the use of sulphanilamide. The formation of a localized peritoneal or cellulitic abscess calls for surgical drainage.

**General Peritonitis.**—Since the introduction of sulphanilamide the incidence and, according to most authorities, the mortality rate of general peritonitis have been considerably reduced. Other valuable aids to treatment are opiates, continuous intravenous therapy and the indwelling duodenal tube. In established general peritonitis—as distinct from spreading peritonitis—the outlook is extremely grave, despite every measure employed; and it is in these cases that I still subscribe to the value of laparotomy and drainage. I am sure that at times this measure swings the balance towards recovery. The lower part of the abdomen and both flanks are drained under local anaesthesia without the patient's being disturbed in her bed.

**Septicemia.**—For anaerobic streptococcal septicemia there is no specific chemotherapy. The best results appear to follow repeated blood transfusions, the aim being to raise the patient's haemoglobin level to between 90% and 100%. Ligation of the thrombosed pelvic veins has been performed, but without reduction in the mortality rate, and today it is rarely practised.

**Irreparable Infection of the Uterus.**—Irreparable infection of the uterus is fortunately rare; but when it is present hysterectomy is necessary. The indications are a grossly infected uterus at Caesarean section, *Clostridium welchii* infection of the uterine muscle, gangrene of the uterus from any cause, and infected fibromyomata.

**Infections with Clostridium Welchii.**—Infections with *Clostridium welchii* are mentioned here because of their special requirements. In addition to the giving of sulphapyridine, their treatment demands removal of the primary focus (fetus, decidua or uterus), massive doses of *Clostridium welchii* antitoxin, intensive hydrotherapy, the administration of alkalis and, commonly, blood transfusions.

#### References.

<sup>10</sup> L. Colebrook: "The Prevention of Puerperal Sepsis", *The Journal of Obstetrics and Gynaecology of the British Empire*, Volume XLIII, Number 4, August, 1936, page 691.

<sup>11</sup> P. Fildes: "A Rational Approach to Research in Chemotherapy", *The Lancet*, Volume I, May 25, 1940, page 955.

<sup>12</sup> E. E. Osgood, J. Joski and I. E. Brownlee: "The Superiority of Neoarsphenamine and Sulphathiazol in the Therapy of Staphylococcus Aureus Infections in Marrow Cultures", *Surgery, Gynecology and Obstetrics*, Volume LXXI, October, 1940, page 445.

#### REPORT ON CEREBRO-SPINAL MENINGITIS.<sup>1</sup>

By LIEUTENANT-COLONEL M. J. HOLMES, D.S.O.,  
Senior Medical Officer, Commonwealth Department of  
Health; Director of Hygiene, Army Headquarters.

CEREBRO-SPINAL meningitis has given evidence of more than normal activity in Australia during 1940. This was to be expected in view of the civil disturbance associated with the war and with military training. As somewhat similar conditions existed in 1915-1916 when cerebro-spinal meningitis reached very serious epidemic proportions in Australia, it would be helpful to consider the present situation in the light of experience of the 1915-1916 epidemics.

<sup>1</sup> This paper appeared in the report of the ninth session of the National Health and Medical Research Council, and is published by permission of the Chairman, Dr. J. H. L. Cumpston.

In the latter part of 1915 many of the military camps in all the States of Australia experienced a sudden and severe epidemic of cerebro-spinal meningitis. This disease was at the same time present in epidemic form in the civilian community. There has been a strong tendency to accept the conclusion that the disease broke out first in the military camps, and that the civilian epidemic followed, and resulted from the camp epidemics. Thus Calov in *THE MEDICAL JOURNAL OF AUSTRALIA* of July 20, 1940, page 53, in discussing cerebro-spinal meningitis in Australia states: "The epidemic in the civil population during the Great War probably arose in the military camps." Again (page 55): "Overcrowded military establishments in time of war must be regarded as the main source of all widespread epidemics." These statements require modification.

In 1915, when, for example, in Victoria the first camp epidemics occurred in August at Seymour and Flemington, already at least fifty cases of cerebro-spinal meningitis had been reported over several months in Victoria, not only from the metropolitan area, but from districts as widely separated as Ballarat, Bendigo, Healesville, Mooroopna and Drouin. Cerebro-spinal meningitis was not at that time notifiable, otherwise more cases might have been recorded from the civil community. The point is that the disease was widespread over a great part of Victoria before the camp epidemics occurred. The sequence appears to have been that the disease had reached abnormal proportions in the civil community during the earlier part of 1915. Sporadic cases appeared in Seymour camp. There were two suspected cases in May. During June and July cases occurred in the camp at an average of about one per week.

In August there was a great recruiting boom to provide reinforcements to make good losses at Gallipoli. The camps at this time were crowded to the utmost limit with new recruits, in wintry weather, when upper respiratory tract infections were raging both in and out of camp. These new recruits left their sheltered home environment to enter rough camps where they were subjected to unaccustomed exposure under adverse climatic conditions,

September as the susceptibles were used up. Many other camps in Australia had a somewhat similar experience at this time (August and September, 1915) and for the same reasons.

The occurrence of these acute camp epidemics naturally had its effect in increasing the severity of the epidemic already existing in the civil community; but whereas the camp epidemics quickly subsided, the disease continued at a fairly high level in the civil community, falling gradually through the following summer. This is illustrated in Figure II; this graph shows the incidence in military camps and in the civil community in Victoria during 1915 and 1916.

It will be noted that in 1916 a rise in the incidence of cerebro-spinal meningitis in the civil community occurred prior to the reappearance of cases in the military camps. The camp epidemics were again short and sharp. (See Figure III, Bendigo camp, 1916.) The incidence in the civil community, on the contrary, continued to rise after the military epidemic had subsided.

The experience in the other States was in general similar to that in Victoria.

It would appear that evidence of rising incidence of cerebro-spinal meningitis is likely to be found first in the civil community. This may be accompanied or followed by the appearance of sporadic cases in the camps. The occurrence or otherwise of a camp epidemic depends largely on circumstances within the camp. As will be shown later, the experience of 1940 has been in accord with these principles.

As the first indications of rising incidence of cerebro-spinal meningitis are likely to appear in the civil community, it follows that close cooperation between the defence services and civil health services is essential, since it gives to the defence services information regarding the behaviour of the disease in the community which enables precautions to be taken in good time in the camps. Such information also facilitates the issuing of orders enforcing appropriate action in the camps in relation to training aspects, as will be explained later.

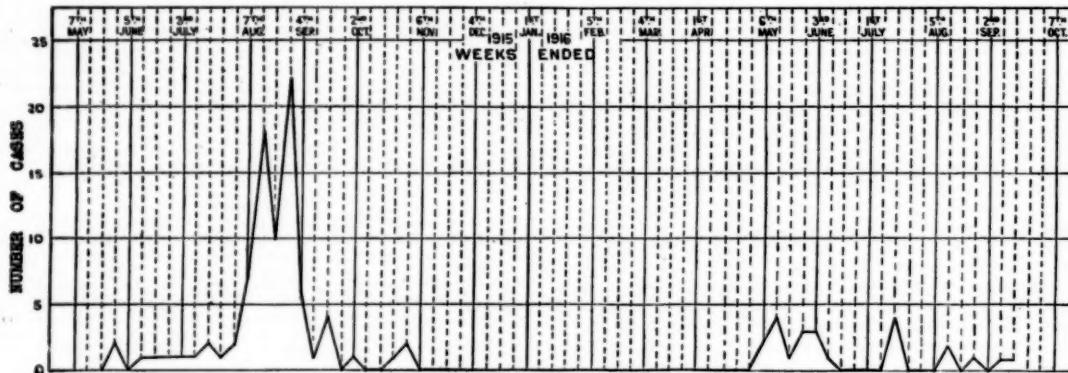


FIGURE 1.

and to heavy fatigue in training, with their vitality temporarily lowered by these adverse conditions and by the effects of inoculation and vaccination, and also by the severe upper respiratory tract infections and other illnesses so readily contracted at first under camp conditions. In these camps sporadic cases of cerebro-spinal meningitis had been occurring for some weeks, and with the influx of large numbers of recruits causing gross overcrowding under the conditions described, it was no wonder that cerebro-spinal meningitis suddenly assumed serious epidemic proportions. In the Seymour and Flemington camps alone 80 cases of cerebro-spinal meningitis occurred within three weeks of the commencement of the epidemic. The course of the disease in the Seymour camp is shown graphically in Figure I. It will be noted that the incidence of cases quickly fell during

#### Cerebro-Spinal Meningitis in Camps.

The experience in Australia during the last war was that an epidemic rise in a camp was usually preceded by a fairly long period during which single cases occurred at intervals of anything between a week and a month. Thus at Seymour camp in 1915 two suspected cases occurred in May. Then there was an average of one case per week through June and July, followed by a sudden heavy epidemic in August. Similarly at Liverpool (New South Wales), two cases occurred in March, 1915, one in June, one in July, followed by the August epidemic.

At Enoggera camp (Brisbane), one case occurred in April, 1915, one in June, and the epidemic in August.

Usually, therefore, the occurrence of sporadic cases over a period gives the cue for energetic precautionary and control measures in the camp. A change to epidemic

proportions may result from a number of factors, the chief of which appear to be (a) excessive overcrowding, (b) influx of a large number of unsalted recruits into a camp in which infection is present, (c) a high rate of upper respiratory tract infection with persistent coughing.

I, a naso-pharyngeal infection, followed in a number of cases by Stage II, bacteraemia, which in turn may be followed by Stage III, metastatic invasion of the meninges. In view of this theory, the association of the cerebro-spinal meningitis epidemic with a prevalence of upper

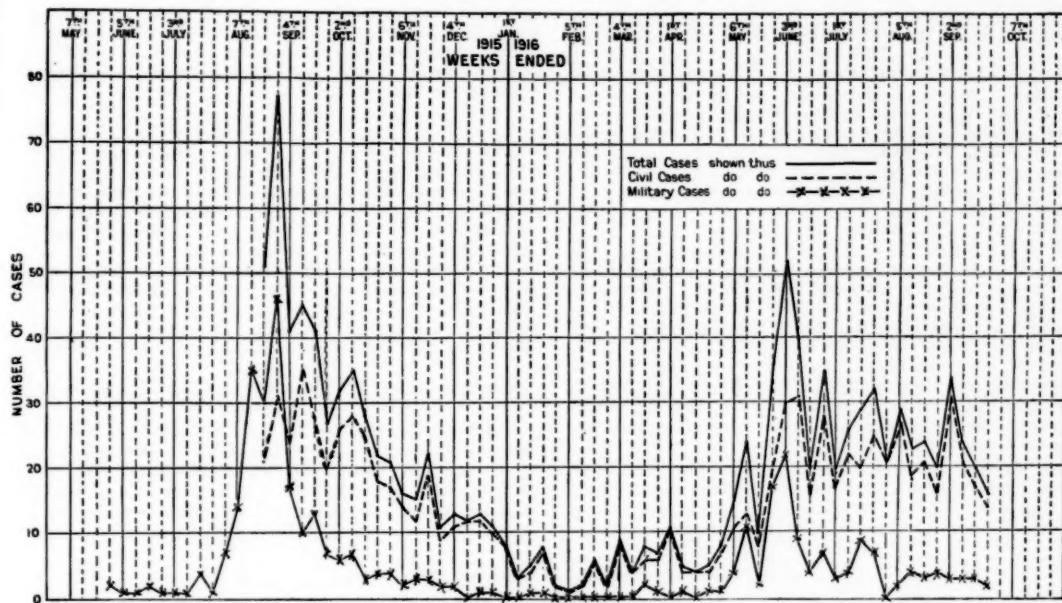


FIGURE II.

All these factors were associated in a great degree with the camp epidemics of 1915-1916.

In 1915 and 1916 severe upper respiratory tract infection, known at the time as "influenza", was very prevalent in the camps for several weeks prior to the occurrence of the camp cerebro-spinal meningitis epidemics. As the wave of so-called influenza reached its peak in a camp, the

respiratory tract infection causing persistent coughing is significant. Even though the upper respiratory tract infection itself may not be due to the meningococcus or to a Gram-negative diplococcus, the persistent universal

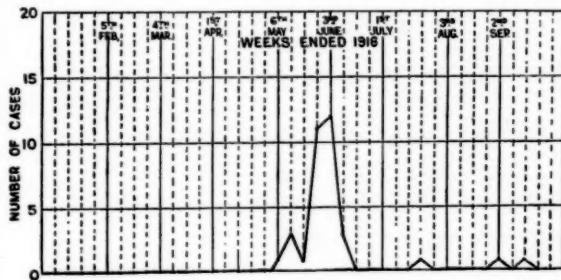


FIGURE III.

cerebro-spinal meningitis epidemic commenced, and when the "influenza" epidemic showed evidence of waning the cerebro-spinal meningitis cases ceased to appear (see Figure IV, Seymour camp, 1915). A great deal of swabbing was done, and it was found that a Gram-negative diplococcus was the predominant organism in the naso-pharyngeal swabs taken in a very large proportion of these so-called "influenza" cases.

Difficulties in connexion with the culture and typing of meningococci prevented the obtaining of any conclusive evidence as to the proportion of cases in which the meningococcus could be isolated from the Gram-negative diplococci present as the predominating organisms in the naso-pharynx.

The accepted theory recognizes three stages in the development of meningococcal meningitis, namely: Stage

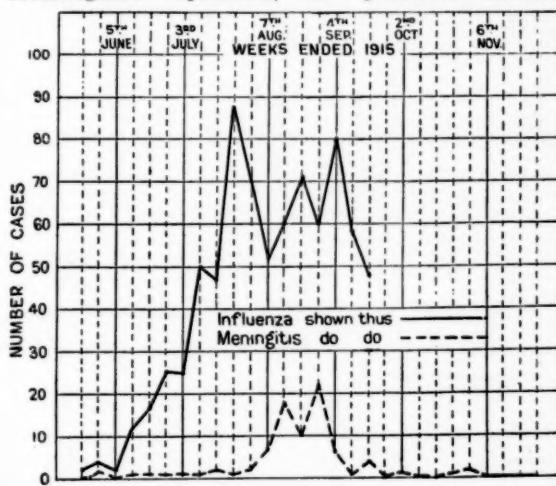


FIGURE IV.

Meningitis cases were those actually diagnosed in camp. "Influenza" cases were cases of febricula, bronchitis, pharyngitis and tonsillitis, the patients being transferred to the base hospital; they did not include cases of suspected meningitis, or any cases in which the patients were treated in camp lines or clearing hospitals or sent home on leave.

coughing accompanying the epidemic of upper respiratory tract infection facilitates the spread of infection from existing carriers of meningococci. Consequently, the prevalence of upper respiratory tract infection in a camp,

no matter what its causative organism, should be a signal for constant watchfulness for any sign or symptom which might possibly be indicative of cerebro-spinal meningitis. The catarrhal infection itself may have the effect of temporarily reducing resistance to invasion by the meningococcus and to the development of meningitis.

*Recruits in Relation to Cerebro-Spinal Meningitis in Camps.*

In 1915-1916 it was found that a large influx of recruits into a camp in which an occasional case of cerebro-spinal meningitis was occurring was likely to be followed by an increased prevalence of the disease and not infrequently by a smart epidemic. The experience of the camps at Seymour and Flemington in Victoria in 1915, in which heavy over-crowding with recruits was immediately followed by the occurrence of more than eighty cases of cerebro-spinal meningitis within three weeks, is suggestive. At the Base Hospital in Melbourne at this time an inquiry was made into the length of time cerebro-spinal meningitis patients had been under training in camp. In nineteen consecutive cases of cerebro-spinal meningitis, the patients having been admitted to the hospital, it was found that in six cases the patients had been one week in camp following enlistment, in four cases they had been two weeks in camp, in five cases they had been three weeks in camp and in three cases they had been four weeks in camp; that is, eighteen out of the nineteen were recruits of less than one month's standing.

I have, earlier in this paper, referred to the risks operating rendering the recruits apparently more liable to the development of cerebro-spinal meningitis. At

Enoggera the step was taken of receiving recruits into a section of the camp where they were held for two or three weeks for modified training before being drafted to the main training camp. The records show that of nine cases of cerebro-spinal meningitis occurring at Enoggera camp between June and September, 1916, seven occurred in the recruits' section. This would appear to indicate that the recruits brought their own infection with them. Under camp conditions favourable to spread of infection, the occurrence of actual cases of cerebro-spinal meningitis resulted in the camp.

*Discussion.*

This summary of the experience drawn from the 1915-1916 outbreak has been given because the experience has been very useful in determining the lines of approach to the problem now that the 1915-1916 conditions are being reproduced in Australia, at least in part. I now pass to consideration of the problems of 1940.

The occurrence of the disease in the civil community and in the military camps will be considered separately and will be followed by a commentary on the relationship between the civil and military developments, and by a review of the measures taken to control the disease.

*Cerebro-Spinal Meningitis in the Civil Community in 1940.*

Table I, showing the monthly incidence of cerebro-spinal meningitis in the several States up to the end of October, 1940, may be considered in conjunction with Table II, showing the seasonal distribution of cases. It will be noted that normally cases of cerebro-spinal meningitis

TABLE I.  
Case Incidence of Cerebro-spinal Meningitis: Australia, 1940.

Month.	New South Wales.			Victoria.			Queensland.			South Australia.			Western Australia.			Tasmania.			Australian Capital Territory.			Northern Territory.			Total			
	C. <sup>1</sup>	M. <sup>2</sup>	T. <sup>3</sup>	C.	M.	T.	C.	M.	T.	C.	M.	T.	C.	M.	T.	C.	M.	T.	C.	M.	T.	C.	M.	T.	C.	M.	T.	
January	3	—	3	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	3
February	1	—	3	1	2	—	2	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	—	4	
March	3	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	3	
April	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	
May	3	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	—	4	
June	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	2	
July	4	1	5	2	—	—	2	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	7	3	10	
August	8	3	11	10	—	—	10	2	—	20	1	1	2	—	—	3	4	7	1	—	1	—	—	24	7	31		
September	7	—	7	20	—	—	20	1	—	2	—	—	—	—	—	16	5	21	—	—	—	—	—	44	6	50		
October	1	1	2	7	7	14	—	—	—	—	—	—	—	—	—	23	3	26	—	—	—	—	—	31	11	42		
Total to end of October	30	5	35	42	7	49	4	2	6	—	—	—	43	12	55	4	1	5	—	—	—	—	—	123	27	150		

<sup>1</sup> C. = Civilian. <sup>2</sup> M. = Military, including Army and Air Force. <sup>3</sup> T. = Total.

TABLE II.  
Seasonal Distribution of Cerebro-spinal Meningitis Weekly Notifications: Australia.

Corresponding Four Weeks Ended.	New South Wales.			Victoria.			Queensland.			South Australia.		
	Mean 1920 to 1939.	1940.	Mean 1920 to 1939.	1940.	Mean 1920 to 1939.	1940.	Mean 1920 to 1939.	1940.	Mean 1920 to 1939.	1940.	Mean 1920 to 1939.	1940.
January 31	1.9	3	1.0	—	0.4	—	0.2	—	0.2	—	—	—
February 28	2.3	1	1.5	2	0.7	1	0.4	—	0.4	—	—	—
March 27	1.8	3	1.3	—	1.4	—	0.3	—	0.3	—	—	—
April 24	1.6	—	0.9	—	1.7	—	0.2	—	0.3	—	—	—
May 22	1.6	2	1.5	—	1.0	—	0.3	—	0.3	—	—	—
June 19	2.2	1	1.5	1	0.6	—	0.3	—	0.3	—	—	—
July 17	2.1	3	1.7	1	0.5	1	0.3	—	0.3	—	—	—
August 14	2.9	9	1.7	4	1.2	2	0.2	—	0.2	—	—	—
September 11	2.6	7	1.6	20	1.1	1	0.7	—	0.7	—	—	—
October 7	3.5	4	1.4	14	0.6	1	0.4	—	0.4	—	—	—
November 6	2.0	—	1.3	—	0.5	—	0.2	—	0.2	—	—	—
December 4	2.1	—	0.9	—	0.7	—	0.3	—	0.3	—	—	—
January 1	1.8	—	1.1	—	0.6	—	0.1	—	0.1	—	—	—
Corrected annual total	28.0	—	19.0	—	10.0	—	4.0	—	4.0	—	—	—

are few in number and are fairly evenly scattered over the year. In none of the States was there any significant departure from the normal weekly incidence up to July, 1940. In most States the incidence was below normal.

In New South Wales, the disease began to show unusual activity towards the end of July, reaching a maximum in August and early September and then gradually declining.

In Victoria the incidence of the disease was lower than usual up to the end of July. About the middle of August evidence of increasing activity appeared, which became pronounced in September, when twenty cases were notified. Signs of declining incidence, however, appeared early in October.

In Western Australia the figures show a rising incidence towards the end of August, which became pronounced in September and has persisted through October. It is understood, however, that a number of cases occurred in the civil community prior to August, but were not notified.

In none of the other States has there been any departure from the normal incidence during 1940.

An attempt was made to ascertain whether the increased incidence of cerebro-spinal meningitis in New South Wales, Victoria and Western Australia subsequent to July, 1940, was scattered over the State or was mainly confined to metropolitan areas. Table III shows that the increase has affected both metropolitan areas and country districts, and the proportion between metropolitan and country cases has not been much disturbed. In Western Australia, activity of the disease in and around Perth and Fremantle appears to have been a feature of the outbreak.

#### Age Group: Distribution of Cerebro-Spinal Meningitis.

It was thought that the rising incidence of cerebro-spinal meningitis might possibly be associated with a definite change in the age group distribution of cases—that is, that there might be a tendency for adults to be attacked in higher proportion, and that such an alteration in the behaviour of the disease might point to an impending epidemic. Only in New South Wales were complete figures available for comparing the age group distribution in 1940 with that of previous years.

Table IV shows that, generally speaking, in the period 1931 to 1939 between 70% and 90% of the patients in New South Wales were under the age of fifteen years. In 1940 this proportion remained unchanged until July. Then, when the disease became more active, the proportion of patients aged over fifteen years rose appreciably; this indicated that the increased incidence was affecting age groups over fifteen years more than age groups under fifteen years.

In Victoria, figures are not available prior to 1939. Table V shows that the proportion of patients aged under fifteen years was much lower than in New South Wales both in 1939 and in 1940, and the increased activity of the disease in Victoria in September and October, 1940, has not been associated with any significant alteration in the age groups attacked.

It cannot be concluded that there has been any significant alteration in the behaviour of the disease in respect of the age groups attacked.

TABLE III.  
Distribution of Cerebro-spinal Meningitis in Metropolitan and Country Areas of Australia.

Year.	New South Wales.				Victoria.			Queensland.			South Australia.			Western Australia.			Tasmania.			Australia.				
	M. <sup>1</sup>	HR. <sup>2</sup>	R. <sup>3</sup>	T. <sup>4</sup>	M.	R.	T.	M.	R.	T.	M.	R.	T.	M.	R.	T.	M.	R.	T.	M.	R.	T.		
1931	..	17	4	0	30	12	11	23	3	4	7	4	2	6	1	—	1	1	2	3	38	32	70	
1932	..	23	4	16	43	16	8	24	1	8	9	—	—	—	5	—	5	—	—	45	36	81		
1933	..	16	—	8	24	5	11	16	—	4	4	1	2	3	1	—	3	3	2	27	28	55		
1934	..	14	5	10	29	8	16	24	1	2	3	1	1	1	2	—	2	4	—	26	36	62		
1935	..	15	—	14	29	10	8	18	—	1	1	3	—	—	2	—	4	1	3	31	28	59		
1936	..	7	1	3	11	11	4	15	1	2	3	2	—	—	1	—	1	2	12	4	23	13	36	
1937	..	7	1	9	17	7	8	15	2	2	4	1	1	2	—	—	—	—	—	17	21	38	—	
1938	..	15	1	6	22	10	8	18	—	2	2	—	—	—	—	—	—	—	—	2	3	27	18	45
1939	..	12	5	5	22	8	6	14	1	5	6	1	—	1	—	—	—	—	—	2	2	22	23	45
Total 1931-1939	..	126	21	80	227	87	80	167	9	30	39	13	6	19	12	6	18	9	12	21	256	235	491	
Annual average 1931-1939	..	14	2	9	25	10	9	19	1	3	4	1	—	2	1	—	2	1	1	2	28	26	54	
1940 (to October 12)	..	21	2	10	33	22	21	43	2	4	6	—	—	—	28	11	39	2	3	5	75	51	126	

<sup>1</sup> M. = Metropolitan. <sup>2</sup> H.R. = Hunter River District. <sup>3</sup> R. = Rest of State. <sup>4</sup> T. = Total.

TABLE IV.  
Age Distribution of Cerebro-spinal Meningitis Cases in New South Wales.

Age Group.	1931.	1932.	1933.	1934.	1935.	1936.	1937.	1938.	1939.	Dates (1940).	
										June 30, 1940, to October 12, 1940.	July 1, 1940, to October 12, 1940.
Under 1 year	..	6	5	5	4	4	2	3	1	6	4
1 to 4 years	..	9	21	11	11	6	3	5	6	7	1
5 to 14 years	..	9	12	4	9	10	4	5	4	3	3
15 years and over	..	6	5	4	5	9	2	4	11	5	13
Total .. .. ..	90	43	24	29	29	11	17	22	22	11	21
Percentage under 15 years	80	88	84	83	70	82	74	50	77	82	38

TABLE V.  
*Age Distribution of Cerebro-spinal Meningitis Cases, Victoria, 1939-1940.*

Age Group.	1939.	1940, up to October 12.
Under 1 year	5	2
1 to 4 years	3	8
5 to 14 years	—	9
15 years and over	6	24
Total	14	43
Percentage under 15 years	43	44

TABLE VI.  
*Age Distribution of Cerebro-spinal Meningitis Cases in Western Australia, 1940.*

Age Group.	1940, up to October 12.
Under 1 year	2
1 to 15 years	13
15 years and over	22
Total	37
Percentage under 15 years	40

#### Cerebro-Spinal Meningitis in the Military Forces.

Up to the end of October, 1940, 28 cases of cerebro-spinal meningitis had occurred in the defence services; in the Army there were 24 cases, in the Air Force there were three cases and in the Navy there was one case.

These cases were distributed over five States. The monthly incidence is shown in Table I.

In New South Wales five cases have occurred; four of these occurred in July and August, and one at the end of October.

In Victoria there were no cases up to the end of September, but seven cases have occurred during October, including two cases in the Air Force.

In Western Australia cases commenced to occur in the latter part of August and still continue in one camp. There have been twelve cases in all in this State, including one in the Air Force and one on a local naval vessel stationed at Fremantle.

Queensland has had three cases, one in December, 1939, one in July and one in September, 1940.

Tasmania has had one case, in July, 1940.

#### Distribution of Cerebro-Spinal Meningitis in the Camps et cetera.

Twenty-eight cases have occurred in sixteen different camps. Northam camp (Western Australia) had eight cases in ten weeks (August 17 to October 31). Darley camp (Victoria) had three cases between October 1 and 3. Redbank camp (Queensland) had two cases, in December, 1939, and July, 1940. Ingleburn camp (New South Wales) had two cases in August (the interval between was eighteen days). Showground camp (Sydney) had two cases, one in July and one in October, 1940 (the interval between cases was 94 days). The remaining eleven cases occurred in as many camps (one case only per camp).

With the exception of Northam camp (Western Australia) the cases in camps have been sporadic. In only one instance has more than one case occurred in any military unit. This one exception was an infantry battalion at Northam, in which two cases occurred at an interval of six days, but without known contact between the patients.

It will be seen that there has been little likelihood of association between cases, since with the one exception mentioned all have occurred in separate military units.

Northam camp, Western Australia, provides the only instance of infection persisting in a camp. Here the time spacing of cases was as follows:

Date.	Interval between Cases.
August 18	—
August 22	4 days
September 4	13 days
September 8	4 days
September 15	7 days
September 20	5 days
October 7	17 days
October 15	8 days

This camp, which accommodates 4,000 men, is the main Australian Imperial Force training camp in Western Australia. It has consequently always been necessary to keep the camp filled to the limit of its accommodation. Recruits could not be diverted to other camps for training. It has not been possible to attain the ideal of avoiding considerable influx of new population into the camp while cases of cerebro-spinal meningitis continue to occur; but the outflow of trained men and influx of recruits have been conducted gradually and with great care, and violent changes have been avoided. With the unavoidable periodic influx of new recruits into a camp in which infection is obviously active, the persistence of cases is to be expected, but the interval between cases shows that the position is under control. Careful attention has been given to the avoidance of overcrowding, and recruits are held and trained for a time in a separate section of the camp. Other precautions taken will be mentioned later under the general discussion on control measures.

In view of the experience in 1915-1916 pointing to a higher incidence of cerebro-spinal meningitis in recruits than in men with longer training, information was sought from all camps affected regarding the length of time between the commencement of training and the development of the disease. This information has been obtained in respect of 20 of the 24 cases from military camps and shows the following length of service:

2 weeks and under	2 cases
3 weeks	2 cases
4 weeks	1 case
6 weeks	5 cases
3 months	5 cases
4 months	3 cases
5 months	1 case
6 months	1 case

It will be noted that half the patients had been six weeks or less in camp when they developed the disease.

#### Commentary.

Except that notifications of cerebro-spinal meningitis in New South Wales were slightly above the average in the first half of 1940, the incidence of notified cases in Australia showed no departure from normal up to July. In July the incidence began to rise in New South Wales. In August this increase in New South Wales became more pronounced, and a definite rise in incidence occurred in Victoria and Western Australia, which became more pronounced in September.

This increase in civil incidence was accompanied by sporadic cases in military camps in New South Wales and Western Australia, but not at first in Victoria. In September the incidence in the civil community increased, but in spite of this no further case occurred in camps in New South Wales, and although there were twenty cases in the civil community in Victoria during this month, no case occurred in the camps in this State until the following month, October.

It will be noted that in New South Wales and Western Australia sporadic cases in camp accompanied but did not precede the rise in notifications in the civil community. In Victoria the pronounced rise in incidence in the civil community in August, followed by a still more pronounced rise in September (thirty cases were notified in these two

months) was not accompanied by any appearance of the disease in the military camps. That the disease would appear in the camps was anticipated, and its appearance was consequently watched for carefully, but it was not till the beginning of October that the first military case was notified.

The assumption that the disease would first show evidence of increased activity in the civil population before appearing in the camps was justified. Cooperation has been arranged with the State departments of health, and notifications in all States were carefully followed. As soon as evidence of increased activity in the civilian population was obtained, instructions were reiterated to all military commands regarding action to be taken on the appearance of a case in the camp. I shall refer to these instructions later; but it is to be noted that in eleven of the camps attacked only one case of cerebro-spinal meningitis occurred. Of the five camps in which more than one case appeared, three had two cases only with a long interval between cases, one had three cases, and one had eight cases.

This experience supports the contention that the camps have not been a main factor or even a large factor in accounting for increased prevalence in the civil community. Rather, the camp cases have been a natural concomitant of the civilian cases. If the carrier rate in the civil community is sufficiently high to cause an abnormally high incidence of cases in the community, then it is natural that the camps fed from the community will also have a raised carrier rate. Conditions of camp life are more favourable to spread of infection than those of civil life, and therefore the appearance of a case in camp is the more likely to be followed by further cases. Unremitting watchfulness and care are necessary in every camp into which the infection is introduced, so as to prevent the development of a camp epidemic which in its turn would react on the civil community and lead to the establishment of a vicious circle.

Although full epidemiological inquiry is made into each military case and the information is forwarded to the Director-General of Medical Services at Army Headquarters, very little evidence is forthcoming regarding association between case and case. Nevertheless contacts, and especially close contacts of cerebro-spinal meningitis patients in camp, are kept under observation, though not isolated unless for special reasons. Cooperation with the State departments of health has been maintained with a view not only to the mutual understanding of the developments in both spheres, but in order to facilitate any epidemiological investigation which may be thought necessary. Immediately on the occurrence of a case in a camp the local health authority and the State department of health are notified. Should the patient have very recently been staying at a civil address, as, for example, while on leave from camp, the State department of health is advised of the fact and of the address, together with any other information of epidemiological interest. This enables the State department of health to make such inquiry as it may desire at the civilian end, in order to ascertain, for example, whether any suspicious illness has occurred at the address in question, or whether contacts need attention there.

Should any soldier develop cerebro-spinal meningitis when away from camp, the State department of health at once advises the Deputy Director of Medical Services of the Command. When a case occurs in the civil community and the State health authorities find that any soldier has been a close contact, the Deputy Director of Medical Services is at once advised so that the soldier may be placed under observation.

These measures of cooperation are strengthened by direct personal cooperation between the permanent head of the State department of health, the Assistant Director of Hygiene in the Command or Military District and the Director of Hygiene at Army Headquarters.

#### *Bacteriological Examination: The Taking of Swabs from Contacts.*

The taking of swabs from contacts is not carried out as a routine measure in the military camps in which cerebro-

spinal meningitis has occurred. In view of the fact that culture and typing of the meningococcus require much experience and great care, are costly in time and material, and require special facilities and equipment which are not always available, the routine employment of these measures for the detection of carriers is not justified. Occasions will arise when bacteriological examination of naso-pharyngeal swabs will give important practical information. When, for example, in a camp there is a prevalence of upper respiratory tract infection, the examination of naso-pharyngeal swabs from a number of patients with catarrhal symptoms will assist in assessing the risk of a cerebro-spinal meningitis outbreak in the camp and so help in determining the scope of the precautionary measures to be adopted. In general the taking of swabs and the bacteriological examination of patients with catarrhal symptoms and of contacts is carried out at the discretion of the Deputy Director of Medical Services or of the pathologist, and not as a routine measure for the detection of carriers. In the case of a patient or suspect, full bacteriological investigation is immediately carried out, including the taking of a naso-pharyngeal swab, lumbar puncture and the culture and typing of the organisms from both sources. Blood culture is also attempted when it is considered desirable. Tests for concentration of sulphapyridine or sulphanilamide in the blood and the cerebro-spinal fluid are carried out, and the blood is examined on the third day and again later on for variation in the leucocyte count and evidence of agranulocytosis.

#### *Control of Cerebro-Spinal Meningitis in Camps.*

Briefly the action taken on the occurrence of a case or suspicious case of cerebro-spinal meningitis in a camp is as follows:

1. The senior medical officer of the camp immediately notifies the Deputy Director of Medical Services, who at once advises the Director-General of Medical Services by telegram. Telegraphic advice to the Director-General of Medical Services is followed as soon as practicable by full epidemiological details and information regarding action to clinch the diagnosis and regarding the treatment and progress of the patient. It may be stated that several cases of suspected cerebro-spinal meningitis have turned out to be pneumococcal or streptococcal in origin. As soon as the diagnosis is confirmed, the local health authority and State department of health are notified and advised of any facts of epidemiological importance, including details of close civilian contacts.

2. The patient is isolated immediately in the camp, and treatment with sulphapyridine in full dosage is commenced as soon as swabs have been taken and lumbar puncture has been performed for diagnostic purposes, but no delay is made for confirmation of the diagnosis.

3. The patient is removed to isolation hospital as soon as practicable.

4. Immediate steps are taken to confirm the diagnosis by bacteriological methods. For this purpose, before sulphapyridine is administered, naso-pharyngeal swabs are taken, lumbar puncture is performed and blood culture may be attempted.

5. The close contacts of the patient are put under observation, but are not isolated. At the discretion of the Deputy Director of Medical Services or senior medical officer, naso-pharyngeal swabs may be taken and examined if facilities for the efficient performance of this work are available. Close contacts are those who occupy beds immediately adjacent to the patient, and any others judged by the medical officer to have had close or prolonged contact.

6. If any close contact under observation develops catarrhal or other signs suspicious of a commencing meningococcal infection, he is isolated, and treatment with sulphapyridine is commenced at once; the result of examination of the naso-pharyngeal swabs is not awaited.

7. Careful watch is maintained in the unit and in the camp generally to detect clinically suspicious cases at the earliest possible moment. Men suffering from catarrh are specially watched.

8. Army instructions for the relief of overcrowding in camps have already been referred to. Additional accommodation in camps is being provided as rapidly as possible, and recruiting activity has been regulated by the accommodation available. In any camp where cerebro-spinal meningitis occurs, everything possible is done to overcome any overcrowding which may exist. Strict attention is paid to the maintenance of ventilation in the sleeping and other quarters, and to the prevention of undue crowding together in recreation huts *et cetera*. The men receive instruction in personal hygiene in relation to droplet infection.

9. Action is taken in relation to cleanliness of tents and huts. In the tents the floor boards are moved periodically into the sunlight and tent sides are turned up to give the sunshine direct access to the space beneath the tents. Huts are thrown open and washed out with disinfectant solution.

10. Leave from the camp is not interfered with, except that close contacts under observation are not given leave until the period of observation has expired. Visitors are not debarred from the camp, but they meet the troops only in the open air.

Should cases of cerebro-spinal meningitis continue to occur in the camp, thus providing evidence of persistent infection, the following additional steps are taken. As far as practicable, entry of recruits to the camp is stopped. If military exigencies will not altogether permit of this, the entry of recruits is reduced to a minimum and is carried out gradually and not in large-scale movements. Recruits are received into a separate portion of the camp. Similarly, movements of troops from the infected camp to other camps, or from other camps to the infected camp, are as far as practicable avoided.

These precautions not only assist in relieving overcrowding in the camp by avoiding or minimizing the influx of troops, but reduce the amount of contact between the new recruits and the infected camp population. In addition, stricter care is exercised in relation to permission for visitors to enter the camp. No children are permitted to visit the camp. Leave of troops from the camp is subject to careful control, and the men are instructed in personal hygiene measures in relation to the conveyance of droplet infections.

In the Northam camp (the only camp in which there has been a persistent recurrence of cerebro-spinal meningitis cases) the step has been taken of actually removing close contacts to a separate isolation block, where they are kept under open-air conditions and not released until two consecutive "negative" swabs are obtained. Close contacts found to harbour meningococci in the naso-pharynx are given a course of sulphapyridine treatment.

Civilian contacts who cannot be satisfactorily isolated and have swabs taken at home have similarly been removed to isolation hospitals and swabs have been taken when necessary. These civilian contacts have been treated.

#### *Treatment.*

Adequate arrangements for isolation and treatment of cerebro-spinal meningitis patients in both the civil population and the defence services are provided in every State. Full instructions regarding treatment have been issued to all military medical officers. Sulphapyridine is employed in full dosage. In fulminant cases the initial dosage is given by the intramuscular and intravenous routes.

Dosage is watched by the estimation of the concentration of sulphapyridine in the blood and cerebro-spinal fluid. A leucocyte count is made on the third day and again later.

#### *Mortality Rate.*

In the 28 cases which have occurred in the defence services there have been three deaths, a mortality rate of approximately 10%. One of the three deaths occurred in a fulminant case within six hours of the commencement of treatment.

### MENINGOCOCCAL INFECTION WITH SPECIAL REFERENCE TO MENINGOCOCCAL SEPTICÆMIA.

By GERALD C. MOSS, M.B. (Melb.), M.R.C.P. (Lond.), F.R.A.C.P.,

*Senior Honorary Physician, Fremantle Hospital,  
Honorary Physician to Out-Patients, Perth  
Hospital, Captain, A.A.M.C.,  
Perth.*

"RESEARCHES of the last decade have shown that the exciting organisms of many infective conditions formerly thought to have a strictly focal distribution, such as typhoid, pneumonia, gonorrhœa and diphtheria, can invade the blood stream. Recognition may then be purely due to bacteriology or to the development of metastatic suppurative processes. Such an invasion of the blood stream is scarcely to be wondered at and even if we cannot prove it, we should infer it far more often than we do." Salomon,<sup>(25)</sup> in describing the first typical case of chronic meningococcal septicæmia, wrote this in 1902, and it is no less true today. Yet the condition is still not so widely recognized as it should be.

It has been asserted recently by Stott and Copeman<sup>(26)</sup> that the clinical diagnosis is simple. Once this would have been regarded as a surprising and dogmatic statement; but there is little doubt that it is justified. There may be, of course, the occasional exception; but in what disease is there not? As is so often the case, it is a question of thinking of the disease. When meningococcal infection is epidemic, as at the present time here, it is to be looked for diligently—and there must be few epidemics of any size in which cases would not be found. The term "meningococcal infection" rather than "meningitis" is used advisedly, for reasons to be mentioned.

Much has been said of the difficulty of obtaining a positive blood culture, a procedure often successful and often not. All agree upon the necessity of scrupulous care in technique, enrichment of media and so forth.<sup>(27)</sup> Every effort should be made to secure a culture. Now, however, in fairness to the patient, too much time should not be spent in repeated trial, quite apart from the fact that it may not be practicable under service and other conditions.

Almost identical descriptions of the rash have now been placed on record many times. Other common features are almost always present, but in themselves would scarcely suffice for diagnosis. More widespread knowledge of the significance of the typical rash in conjunction with the other symptoms will lead to a diagnosis in many cases at the first examination. This is of even more importance than formerly, for sulphapyridine will bring about a speedy cure. Dimson<sup>(28)</sup> in 1938 cured a patient with "M & B 693", and expressed the opinion that it should always be used. He was struck by the rapid effect, compared with the slower response obtained in two cases by Zendel and Greenberg, who used sulphanilamide. Bryan and Mackay<sup>(29)</sup> admit that their patient was much better for some weeks after "M & B 693", although he relapsed. Such a relapse must be very unusual; but much more is known about dosage now than then, and one would like more details about the method adopted. Stott and Copeman's results with the drug surely afford conclusive proof of its efficacy. It is probably not too much to say that this prompt therapeutic response is an additional diagnostic feature. Results with sulphapyridine in meningococcal meningitis have been spectacular here, as elsewhere. It is only to be expected that they would be even better in the slow forms of septicæmia without meningeal localization.

A disease that may entail weeks or months of illness (28 months with death, in one of Kennedy's cases) and may end in meningitis or even meningococcal endocarditis<sup>(30)</sup> with or without other complications,<sup>(31)</sup> can scarcely be called benign in spite of a strong tendency to spontaneous cure.

Lambie's<sup>(23)</sup> case seems to be the only one of subacute or chronic meningococcaemia to have been described in Australia. He mentions Fairley and Stewart's work in 1916; but these authors did not connect the typical rash with a slow septicaemia.

This being so, two cases of my own are reported and mention is made of others seen through the courtesy of a colleague. There is some reason for thinking that more cases are likely to be met with in the later stages of an epidemic, for immunological reasons.

Writers quoted have traced the successive steps by which knowledge of the disease has been assembled. These, perhaps, may once more be summarized. This knowledge begins with the demonstration by Gwyn, in 1899, of meningococci in the cerebro-spinal fluid, blood and synovial fluid in a case of epidemic meningitis with arthropathy (Bloedorn<sup>(24)</sup>). Salomon's case was the first one of true chronic meningococcal septicaemia to be published. Meningitis ensued after two months and the patient recovered; a positive blood culture was obtained. A fulminating case of meningococcaemia without meningitis was reported by Andrewes<sup>(25)</sup> in 1906. Although we are not concerned here with acute fatal cases, it is interesting to note that it was a physician in whose case Andrewes had "the melancholy satisfaction of making a diagnosis".

It is possible to mention only some others whose reports have increased our knowledge of the disease. In Great Britain descriptions have been given by Kennedy<sup>(26)</sup>, Stote<sup>(27)</sup> and Stewart Wallace<sup>(28)</sup>. Kennedy's excellent article with a report of four cases seems to have been largely overlooked. The last of three patients reported by Stewart Wallace was seen by me when I was on a visit to the London Hospital in 1935; a fatal terminal meningitis ensued ten months after the onset of the illness, but at no time could a positive blood culture be obtained.

In Germany, there are the accounts of Friedemann and Deicher<sup>(29)</sup> and of Herrmann and Lifschitz<sup>(30)</sup>. The former writers proposed the name of the "lenta" form of meningococcal sepsis. They point out the similarity of the symptoms and temperature to malaria in their own cases and in two cases of Deycke's. In America, mention may be made of articles by Cecil and Soper<sup>(31)</sup> on cases associated with endocarditis, and by Bloedorn<sup>(24)</sup>, Dock<sup>(32)</sup>, Marlow<sup>(33)</sup>, Graves, Dulaney and Michelson<sup>(34)</sup> and Carbonell and Campbell<sup>(35)</sup>.

Further references are given to Continental literature, from which it appears that slow forms of meningococcaemia were well known, especially in France.

A recent annotation in *The Lancet* draws attention to the importance of diagnosis and treatment<sup>(36)</sup>. Two cases occurring in soldiers will now be described.

1940, he twisted his left knee in stepping from a ferry, and was admitted to the same hospital next day. No detailed account is available of his progress there; but he appeared to have influenza on the day of his readmission. A rubella-like rash was noted on October 15, 1940, and this went away in a few days. He was noted then to have pain and a definite effusion in his right knee, and in addition had pain in both wrist joints sufficiently severe for "Plastine" to be applied. All this time he had irregularly intermittent elevations of temperature. Rises to 101° F. or 102° F. about every three to five days would be followed by intervening periods of slightly elevated, normal or subnormal temperature; later, the apyrexial periods became shorter. Some resemblance to the quartan form of malaria can be seen, although the intermittence was not regular. He had many carious teeth, and two were removed as a possible infective focus.

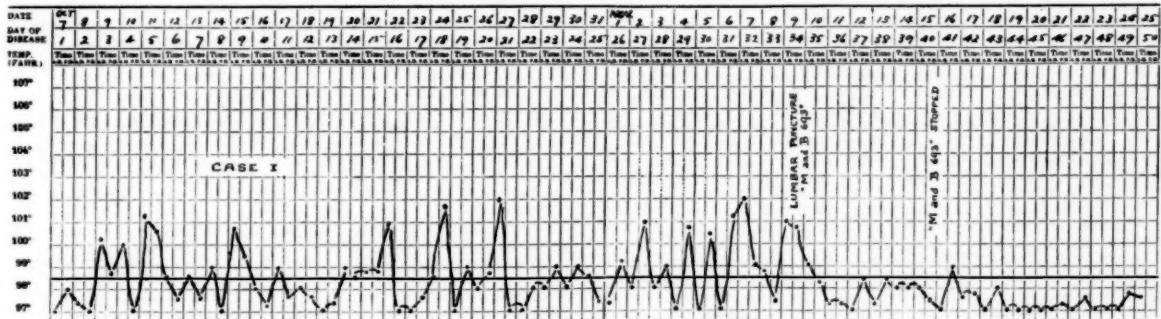
He was first seen in the 110th General Hospital on November 4, 1940. Intermittent pyrexia continued; but there was no pain or swelling in the joints on his admission to hospital; nor was there any obvious rash. He complained of some headache, but felt well. Many carious teeth were present; but otherwise examination revealed no abnormality. The patient denied having had gonorrhoea, and neither examination of a prostatic smear preparation nor the complement fixation test for gonorrhoea revealed the disease.

On November 6, 1940, pyrexia was still present; but the patient had no complaint save of some drowsiness. It was then thought that the rash noted as rubella might have been due to chronic meningococcal septicaemia, and he was thoroughly reexamined. One or two small faint brown macules were seen on the left forearm, but were thought to be of doubtful significance. Otherwise examination revealed no abnormality; no glands were palpable, nor was the spleen to be felt. Arrangements were made for the following investigations: (a) a full blood count, (b) an attempt at culture of meningococci from the blood, (c) an attempt at culture from a sterile specimen of urine. These tests were not carried out until November 8, 1940.

On November 7, 1940, his temperature was 101.6° F.; he had some frontal headache, but no neck stiffness, and Kernig's sign was not elicited. Three pink macules the size of a threepenny piece were seen on the ulnar aspect of the left forearm and one slightly raised papule was found on the volar surface of the same forearm. A macule of about thumb-nail size was seen over the second metacarpophalangeal joint of the left hand. The joint was tender. The macules disappeared on pressure. Three small lesions resembling petechiae and not disappearing on pressure were on the dorsum of the left foot. This was a highly suspicious state of affairs.

On the morning of November 8 he said that his headache was much better and was not worrying him. The skin lesions could no longer be seen. Blood was taken for inoculation into nutrient media. In the evening again he had slight headache with slight neck stiffness. On the afternoon of November 9 he said that his headache was more severe. Definite neck stiffness was present and Kernig's sign was elicited; no rash was present.

Lumbar puncture was performed; the initial pressure of the fluid was 260 millimetres of cerebro-spinal fluid; 35



#### Reports of Cases.

CASE I.—Private N., aged twenty-one years, was admitted to another hospital on September 10, 1940, suffering from acute appendicitis. At operation the same day a slightly inflamed appendix was removed. His progress was satisfactory and his temperature normal for the last few days he was in hospital. After two weeks he was transferred to a convalescent depot, where he felt well. On October 6,

cubic centimetres of opalescent fluid were removed. The final pressure was 100 millimetres of cerebro-spinal fluid. An immediate intramuscular injection of "Sodium Dagenan" (one gramme) was given, and two tablets of "M & B 693" (one gramme) were given by mouth; at 4 p.m. the temperature was 101° F. Further treatment by "M & B 693" was given *secundum artem*; a total dosage of 34 grammes was given in six days. The patient's temperature was

normal at 8 a.m. next day and has been normal since. Progress was uninterrupted except for nausea from the sulphapyridine. The patient was allowed up on the fourteenth day, and at the time of writing is quite well.

The laboratory investigations were undertaken by Captain Michaels. On November 8, 1940, a sterile specimen of urine was found to contain no pus cells; no culture could be obtained and no sugar or albumin was found in the urine. A small Gram-positive diplococcus was grown in culture from the blood. It would seem that the organism was a contaminant. A blood count revealed that the haemoglobin value was 78.5%; the red cells numbered 4,130,000 per cubic millimetre, the colour index was 0.95, and the leucocytes numbered 9,000 per cubic millimetre. A differential leucocyte count revealed 56% polymorphonuclear cells, 36% lymphocytes, and 8% monocytes.

A blood count on the day the sulphapyridine therapy was stopped revealed some leucopenia and granulocytopenia; but the number of leucocytes and of granulocytes had risen a few days later, without special measures. The following are the results of these blood counts. On November 15, 1940, the haemoglobin value was 77%, the red cells numbered 4,130,000 per cubic millimetre, the colour index was 0.94, and the leucocytes numbered 3,800 per cubic millimetre; 38% were polymorphonuclear cells, 60% were lymphocytes and 2% were monocytes. On November 18 the haemoglobin value was 75%, the red cells numbered 3,780,000 per cubic millimetre, the colour index was 1.0, and the leucocytes numbered 6,800 per cubic millimetre; 47% were polymorphonuclear cells, 51% were lymphocytes and 1% were monocytes. Iron was given.

Examination of the cerebro-spinal fluid revealed that the cells numbered 1,660 per cubic millimetre; the globulin content was greatly increased; the total protein content was 160 milligrammes per 100 cubic centimetres. The chloride content was 695 milligrammes per 100 cubic centimetres, and the sugar content was reduced. Cytological examination revealed that about 60% of the cells were lymphocytes and endothelial cells and 40% were polymorphonuclear cells. A pure culture of *Neisseria meningitidis* was obtained.

This, then, was a case of chronic meningococcal septicæmia, lasting at least thirty-three days and terminating in meningitis. A systematic description of the original rash could not be obtained. In the next case both temperature chart and rash were striking, and with the history enabled the diagnosis to be made immediately.

**CASE II.**—Private H., aged twenty-one years, a quarter-caste Maori, was admitted to a camp dressing station on November 9, 1940, with a history of weakness, headache, pain in the limbs, anorexia and shivering attacks of one week's duration. It was noted that his temperature was 101.8° F., his pulse rate was 88 per minute, his respirations numbered 20 per minute, and his tongue was furred; labial herpes was present and there was a rash on the trunk, arms and legs. On November 12, 1940, the rash was fading. On November 14, 1940, a positive reaction was obtained to the Widal test. He was sent to the 110th General Hospital the next day.

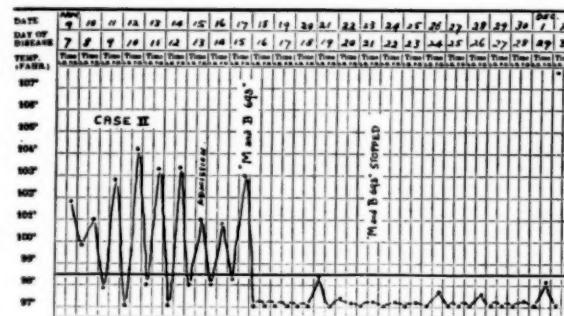
When seen on November 15 he was reading, and neither looked nor felt ill, although inspection of the temperature chart which accompanied him revealed an intermittent pyrexia, ranging from subnormal in the mornings usually to 103° or 104° F. in the evenings.

The patient gave the following history. The onset was sudden, thirteen days previously, with malaise, occasional pain in the arms and legs, anorexia, profuse sweats and frontal headache. The same day he noticed he had an eruption of roundish pink spots about the size of a threepenny piece on the legs, chest and abdomen. In a few days these went away, and he thinks that they mostly diminished in size, leaving small raised red pimples, not much larger than a pin's head. On the legs he thinks that the lesion gradually faded without diminishing in size. A few days later a similar rash appeared on the back of his hands and the back and the front of the forearms. The general symptoms had been present at intervals since; he was also constipated. He had general malaise and chilliness before sweating, but for much of the time felt quite well. He had suffered from measles at the age of five years and had had dengue fever in north-west Australia one year earlier. There was nothing of note in his family history; his father was a half-caste Maori.

Physical examination, except for the obvious rash, revealed no abnormality. There were no palpable glands and the spleen could not be felt; no herpes remained, and there were no meningeal signs. On the back of the hands and both surfaces of the upper limbs was an assortment of vivid pink macules and papules, varying in size from a pin's head to a threepenny piece. They were mostly round or slightly oval, and disappeared completely on pressure.

Similar lesions, generally smaller, were seen over the back, the front of the chest and the abdomen; others of varied size were present on the back and front of the legs and on the dorsum of the feet. A larger oval, pink, slightly raised papule was seen over the dorsal aspect of the second metacarpo-phalangeal joint of his left hand. A similar larger macule was noted on the dorsum of the right foot and the outer aspect of the metatarsal-phalangeal joint of the right great toe. These larger lesions were very like those of *erythema nodosum*, but were only slightly or not at all raised. They disappeared on pressure and were tender, especially those overlying small joints.

Here, then, was a typical case of the slow form of meningococcal septicæmia. The positive response to the Widal test could be accounted for by previous inoculation with "T.A.B." vaccine and could be neglected. As a matter of interest the test was repeated and the patient's blood was found to agglutinate typhoid and paratyphoid strains of a titre of 1 in 80. The following investigations were immediately carried out on November 15, 1940: a blood culture was obtained, a naso-pharyngeal swab was examined for meningococci, and a full blood count was made.



On November 16 his condition was the same. A florid rash was still present; but some spots had faded to faint brown stains. Others were merely less vivid. He had some pain in his right knee and the joint was painful on active and passive movement; but no swelling or effusion was present.

On the evening of November 17 the patient still felt fairly well; but his temperature was 103° F. and he had sweated freely. Many of the skin lesions had faded and there were a few fresh ones. It was thought advisable to terminate the illness as soon as one more sample of blood had been taken for inoculation into nutrient media. This was done, and treatment with "M & B 693" was begun at 10 p.m. It was stopped after he had had a total of 23 grammes in six days.

On November 18, at 8 a.m., the temperature was normal; it remained so for the rest of his stay in hospital. He felt very well. He had again sweated profusely during the night. Most of the skin lesions had faded, leaving a light brown stain; others had disappeared completely. A few small rose-coloured ones were still present. None showed petechiae in the centre. The right knee was no longer painful. On November 19 he felt quite well and had had no further sweating. All that remained of the rash were a few faint brown macules on his forearms and feet. Progress thereafter was uninterrupted. On November 20 a few faint macules were present on the feet only. On November 22 a few faint macules on the feet were seen for the last time. On December 2 the patient was discharged from hospital to his unit for light duty.

A number of laboratory investigations were carried out by Captain C. Fortune. On November 15 examination of a naso-pharyngeal swab revealed *Neisseria meningitidis*. Inoculation of blood into nutrient media produced no growth of organisms. A blood count gave the following information: the haemoglobin value was 85%, the red cells numbered 4,150,000 per cubic millimetre, and the colour index was 1.04; the leucocytes numbered 14,400 per cubic millimetre, 69% being neutrophilic cells, 1% eosinophile cells, 3% old metamyelocytes, 3% monocytes and 24% lymphocytes; no basophilic cells were seen.

On November 17 blood culture yielded a growth of Gram-negative diplococci morphologically resembling *Neisseria meningitidis*; but subculture was impossible. (This happened in Salomon's case, in one of Kennedy's cases and in other cases.) On November 27 a blood count gave the following information: the haemoglobin value was 86%, the red cells numbered 4,390,000 per cubic millimetre, and the colour index

was 1.0; the leucocytes numbered 9,900 per cubic millimetre, 55% being neutrophile cells, 2% eosinophile cells, 1% basophile cells, 2% old metamyelocytes, 5% monocytes, 35% lymphocytes.

On November 30 examination of a naso-pharyngeal swab revealed no *Neisseria meningitidis*.

At no time did he have meningeal signs, so lumbar puncture was not performed.

While attending soldiers suffering from meningitis at the infectious diseases branch of Perth Hospital, I have been enabled to see civilian patients through the courtesy of the medical staff. The following case histories may be briefly quoted.

A woman, aged thirty-six years, was seen on December 6, 1940, on her admission to hospital. Two days before, she had felt as if the sun had struck her in the back of the neck. She began to shiver and her limbs felt stiff and sore. The next day she had an aching pain in the back of the neck and a rash appeared. It is superfluous to describe the rash, as it was a replica of that described in Case II, although not quite so extensive. She had moderate neck stiffness and Kernig's sign was elicited. Lumbar puncture produced 18 cubic centimetres of crystal-clear fluid at a pressure of 200 millimetres of cerebro-spinal fluid. The final pressure was 60 millimetres. She responded immediately to treatment with "M & B 693" and was allowed up in a few days. The cerebro-spinal fluid contained no cells, no increase in globulin and no organisms.

It is interesting to speculate as to when an inflammatory exudate would have appeared in the cerebro-spinal fluid. As likely as not, the meningeal signs would have been transient.

A man, aged fifty years, was convalescent when first seen on November 12, 1940; he was admitted to hospital on November 8, stuporous and with pronounced meningeal signs. Lumbar puncture was performed and an opalescent fluid at 120 millimetres pressure was obtained. In the fluid a few disintegrated polymorphonuclear leucocytes were seen; there was unavoidable delay in its examination. The patient had become ill three weeks before his admission to hospital, with sudden headache, shivering and pain in the legs and back. The pain became worse and was felt especially in the ankles and knees. He sweated profusely at night and felt better in the morning. One week after the onset he noticed a rash; his description of this was quite typical. Frontal headache had become worse five days before his admission to hospital. Up to this time he had carried on his work, with an occasional day in bed. On the day of his admission he was much worse and vomited. The headache was intense. The rash was not seen in hospital; his wife said that it had faded two days before he was admitted.

During this epidemic, there have been several cases associated with petechiae and purpuric rashes, with or without arthropathy and clear cerebro-spinal fluid, in which a rapid response to sulphapyridine therapy has been obtained. Shortage of staff makes it difficult to attempt blood cultures.

McLean and Caffey<sup>(27)</sup> following previous authors, demonstrated meningococci in smears from purpuric lesions in children in 83% of cases in which such lesions were present. In a few of these the cerebro-spinal fluid was normal.

Recently, the tensely swollen knee joint of a patient suffering from meningitis had to be aspirated. Purulent sterile fluid was obtained, but no meningococci, as he had just finished a course of sulphapyridine therapy. However, the organism has been obtained from the synovial fluid also.

#### Signs and Symptoms of Meningococcaemia.

Some description of the signs and symptoms of the subacute and chronic forms of meningococcaemia may now be given. The onset is usually, but not invariably, sudden, with malaise, shivering, pains in the limbs, especially the legs, and headache. The headache is of varying degrees of intensity and may come and go. This is true also of meningeal signs. Shivering may be of any degree, from a chilly feeling to a rigor. The pain in the extremities is not necessarily confined to joints, but in the later stages it frequently is; a joint effusion may or may not be present. With the shivering and rise of temperature there may be a profuse skin action and attendant discomfort, yet the patient may feel quite well with the fall of

temperature. This comparative well-being has impressed most who have seen patients. The temperature chart may have a resemblance to that of malaria, as mentioned; the resemblance is stronger when, as in a few cases, the spleen is palpable. As often as not, the temperature is of the common "septic" type.

It is not proposed to give a list of the conditions which have caused mistakes in diagnosis; most of the authors quoted have supplied these, and reflection will call to mind many possible sources of error. At the present time, patients suffering from brucellosis are being treated with sulphapyridine, and some of these patients have had rashes. It is suggested that when a prompt cure by the drug has been brought about the evidence for the diagnosis should be carefully scrutinized, especially when meningococcal infection is epidemic.

As the rash is so important, it is worthy of a detailed description. Once seen, it is not likely to be forgotten. Needless to say, minor variations exist; but in general it has been found to run true to type. The same basic pattern is almost always present. The lesions consist of pink or red macules and papules of varying size. The size in most cases varies from that of a pin's head to a threepenny piece; a few larger lesions are often present. The smaller ones are roughly circular, the larger ones often oval or longer than they are broad. The large ones are usually tender, especially when situated over bone or joint in the extremities. The limbs are usually most affected; but lesions are commonly seen on the back and chest, sometimes the abdomen and more rarely the face. The colour may be uniform or the centre more vivid. Complete disappearance with pressure is the rule, except in some cases (not in the present ones) in which there is a petechia in the centre. Isolated petechiae may coexist; but this is a much less striking feature than the predominant rash. Tender papules or nodes may be found on the shins. It is easy to see how these may be confused with those of *erythema nodosum* or even of *erythema multiforme*. Reflection will show, however, that many of the characteristics would be most unusual in those conditions. The lesions usually fade in a day or so, some completely; in other cases a faint brown stain persists for some days. At intervals, often corresponding with an access of fever, other lesions may appear. *Herpes labialis* carries as much diagnostic weight as it does in meningitis, and no more.

#### Discussion.

Can all this explain certain features of meningitis that have not been generally understood? There is little doubt that it can. It explains how it is that patients with mild, "ambulant", abortive and intermittent types of meningitis have been able to go about their duties for days or weeks, when actually meningeal implantation has not taken place. These cases are not infrequent in the declining stages of an epidemic.<sup>(28)</sup> The puzzling cases of "meningitis" in which the cerebro-spinal fluid is normal are likewise explained. Therefore we should think in terms of "meningococcus infection" and not in terms of meningitis. This conception is not new; but it should receive wider recognition.<sup>(29)</sup>

Bloedorn<sup>(30)</sup> quotes Herrick's opinion that the cases in which there is a macular rash resembling early chicken-pox or large rose spots are of milder types. Fairley and Stewart<sup>(31)</sup> quote eleven cases associated with macular or erythematous blotches; only one of the patients died. Of 108 patients with a petechial or purpuric rash, 64 died.

Dock's<sup>(32)</sup> analysis revealed that, of 26 serum-treated patients suffering from meningitis following meningococcal septicaemia, only three died.

In a recent case of meningitis the patient had typical large tender papules on the dorsum of the feet, on the ulnar surface of the left forearm and over the styloid process of the left ulna. The left wrist joint was painful and swollen. He had milder arthropathies of both knees and one hip. The illness had been present for three days. It was a mild infection; the cerebro-spinal fluid was opalescent and was under a pressure of only 200 milli-

metres; there were 570 cells per cubic millimetre, the globulin content was greatly increased and a pure culture of *Neisseria meningitidis* was obtained. Joint effusions of identical nature are met with in chronic meningococcal septicaemia without meningitis and in patients found to be suffering from meningitis. Painful joints without effusion are just as common in both phases of meningococcal infection. The arthropathies, the rash and the "meningitis" itself are therefore but incidents in meningococcal infection of the blood stream. If the organism's favourite nidus—the meninges—can escape infection, so also can the skin and joints. The difficulty of obtaining bacteriological proof of this in a given case need not again be stressed. Proof, however, is available even in the case of the roseolar papule.<sup>13</sup>

It seems that the development of the roseolar rash is a sign of good resistance in the patient or of waning vitality of the organism. A waxing of its vitality may kill the patient, as has been shown; but sulphapyridine rapidly decides the issue in his favour.

The fortunately rarer fulminating cases associated with massive haemorrhages in the adrenal glands (the Waterhouse-Friderichsen syndrome) are beyond the scope of this article.<sup>14,15</sup> Experience of these in epidemics is leading to the keeping of a sharper lookout for them.

#### Summary.

The steps by which subacute or chronic meningococcal septicaemia has been shown to be recognizable clinically have been outlined. Diagnostic features are described with special reference to the characteristics of the rash. A failure to obtain organisms by culture methods from the blood is of no significance when typical signs and symptoms are present, and treatment should not be too long withheld.

Two representative cases are described and reference is made to others encountered. The incidence is approximately parallel to that of epidemic meningococcal meningitis in the community; as with frank meningitis, sporadic cases can occur.

The presence of the meningococcus in the blood is to be envisaged primarily; its predilection for joints and skin as well as the meninges should then receive due weight. The heart valves may be affected.

The term "meningococcal infection" suggested by Herrick deserves wider adoption.

There is much evidence that the roseolar rash described is a sign of good omen. Sulphapyridine treatment rapidly cures subacute and chronic meningococcaemia; and if meningeal localization takes place in these cases, the prognosis is even better than the now vastly improved one of meningitis generally.

#### Acknowledgements.

I should like to express thanks to Colonel D. M. McWhae, C.M.G., C.B.E., the Deputy Director of Medical Services, Western Command, for permission to report the cases; and to Dr. I. O. Thorburn, honorary medical officer, infectious diseases branch of Perth Hospital, and Dr. R. le P. Muecke, medical superintendent of Perth Hospital, for giving me access to civilian patients.

I should also like to thank pathologists mentioned for their interest and help.

#### References.

- <sup>14</sup> F. W. Andrewes: "A Case of Acute Meningococcal Septicaemia", *The Lancet*, Volume I, April 23, 1906, page 1172.
- <sup>15</sup> W. E. Bloedorn: "Meningococcus Septicaemia", *The American Journal of the Medical Sciences*, Volume CLXII, 1921, page 881.
- <sup>16</sup> W. A. Bloedorn: *Loco citato*.
- <sup>17</sup> W. A. Bloedorn: *Loco citato*.
- <sup>18</sup> W. Boyd: "The Pathology of Internal Diseases", 1931, page 723.
- <sup>19</sup> M. Bryan and H. Mackay: *Proceedings of the Royal Society of Medicine*, Volume XXXII, September, 1939, page 1371.
- <sup>20</sup> H. M. Butler: "Blood Cultures", 1937, page 194.
- <sup>21</sup> A. Carbonell and E. P. Campbell: "Prolonged Meningococcaemia", *Archives of Internal Medicine*, Volume LXI, 1938, page 648.
- <sup>22</sup> R. L. Cecil and W. B. Soper: "Meningococcus Endocarditis, with Septicaemia", *Archives of Internal Medicine*, Volume VIII, 1911, page 1.
- <sup>23</sup> R. L. Cecil and W. B. Soper: *Loco citato*.
- <sup>24</sup> S. B. Dimson: "Chronic Meningococcal Septicaemia Treated with 2-(p-aminobenzene sulphonamido) Pyridine", *The Lancet*, Volume II, August 20, 1938, page 424.
- <sup>25</sup> W. Dock: "Intermittent Fever of Seven Months' Duration, due to Meningococcaemia", *The Journal of the American Medical Association*, Volume LXXXIII, 1924, page 31.
- <sup>26</sup> W. Dock: *Loco citato*.
- <sup>27</sup> W. Dock: *Loco citato*.
- <sup>28</sup> N. H. Fairley and C. A. Stewart: "Cerebro-Spinal Fever", Commonwealth of Australia Service Publication, Number 2, page 65.
- <sup>29</sup> U. Friedemann and H. Deicher: "Ueber die Lenta-Form der Meningokokkenseptikose", *Deutsche medizinische Wochenschrift*, Volume LII, Number 18, 1926, page 733.
- <sup>30</sup> W. H. Grace, C. V. Harrison and T. B. Davie: "Suprarenal Haemorrhage in Meningococcal Septicaemia", *The Lancet*, Volume II, 1940, page 102.
- <sup>31</sup> W. R. Graves, A. D. Dulaney and L. D. Michelson: "Chronic Meningococcaemia", *The Journal of the American Medical Association*, Volume XCII, 1929, page 1923.
- <sup>32</sup> H. Heimann and S. Feldstein: "Meningococcus Meningitis", 1913, page 104.
- <sup>33</sup> O. Herrmann and M. Lifschitz: "Meningokokkenseptikose (lenta)", *Deutsche medizinische Wochenschrift*, Volume LIV, Number 9, March 2, 1928, page 355.
- <sup>34</sup> J. F. Hughes: "Fulminating Septicaemia Associated with Purpura and Adrenal Haemorrhage", *The British Medical Journal*, Volume II, 1940, page 353.
- <sup>35</sup> J. C. Kennedy: "Meningococcus Septicaemia", *The Journal of the Royal Army Medical Corps*, Volume XLVII, 1926, page 6.
- <sup>36</sup> J. C. Kennedy: *Loco citato*.
- <sup>37</sup> C. G. Lambie: "Persistent Benign Meningococcal Bacteriæmia, with a Report of a Case", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume I, 1938, page 1058.
- <sup>38</sup> "Missed Meningococcal Infections", *The Lancet*, Volume II, 1940, page 48.
- <sup>39</sup> F. W. Marlow, Junior: "Meningococcaemia: Report of a Case, with Recovery", *The Journal of the American Medical Association*, Volume XCII, 1929, page 619.
- <sup>40</sup> S. McLean and J. Caffey: "Endemic Purpural Meningococcus Bacteriæmia in Early Life: the Diagnostic Value of Smears from the Purpural Lesions", *American Journal of Diseases of Children*, Volume XLII, November, 1931, page 1053.
- <sup>41</sup> H. Salomon: "Ueber Meningokokkenseptikose", *Berliner klinische Wochenschrift*, Volume XXXIX, Number 45, 1902, page 1045.
- <sup>42</sup> A. M. Stewart Wallace: "Chronic Meningococcaemia", *The British Medical Journal*, Volume I, 1936, page 931.
- <sup>43</sup> C. L. Stote: "The Clinical Picture Produced by Subacute Meningococcal Septicaemia", *The Lancet*, Volume II, 1929, page 701.
- <sup>44</sup> A. W. Stott and W. S. C. Copeman: "Chronic Meningococcal Septicaemia Associated with an Outbreak of Cerebro-Spinal Fever", *The Lancet*, Volume I, June 22, 1940, page 1116.

#### Notes on Books, Current Journals and New Appliances.

#### REGIONAL ANATOMY.

IN the issue of December 18, 1937, attention was drawn in these pages to the second edition of Sections I to V of E. B. Jamieson's "Illustrations of Regional Anatomy". The third edition has now appeared.<sup>1</sup> Dr. Jamieson is senior demonstrator and lecturer in the department of anatomy of the University of Edinburgh, and this alone gives a certain *imprimatur* to the atlas. The second edition was commended to readers; the third is in certain respects an improvement on the second and will no doubt achieve the same popularity that its predecessor has done. The five sections deal with the central nervous system, the head and neck, the abdomen, the pelvis and the thorax. Sections VI and VII, dealing with the upper and lower extremities, have not yet been issued. The total number of plates in the first five sections is 218; the number of coloured illustrations has been increased and many of the coloured illustrations contain more colours than before. As the printing has been well done, the result of this increase in colour enhances the value of the publication. The names used in the legends are those approved by the Anatomical Society of Birmingham in 1933; but where the names differ radically from the Basle *Nomina Anatomica* ("B.N.A.") these names have been inserted in parentheses. This atlas will be useful to the student in the dissecting room and also to the practising surgeon.

<sup>1</sup> "Illustrations of Regional Anatomy", by E. B. Jamieson, M.D.; published in five sections; Third Edition; 1941. Edinburgh: E. and S. Livingstone. Folio 4to. Price: 36s. 6d. net.

# The Medical Journal of Australia

SATURDAY, MAY 3, 1941.

*All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.*

*References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

*Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.*

## AUSTRALIAN DOCTORS AND THE WAR.

Most people have learned by this time that trained medical graduates have to take a most important part in the conduct of a war—that they have to conserve the health of the troops and that in their treatment of the sick and wounded the first consideration must always be the filling of gaps in the combatant ranks, in other words, the return of men to duty as soon as they are fit. These demands being satisfied, it is the function of the medical personnel to see that those not likely to become fit for duty are returned as far as possible to their former health and strength. What numbers of people, including perhaps some members of the medical profession, do not realize, is that these duties demand large staffs. The size of medical units, whether in Navy, Army or Air Force, is determined by the work that may have to be done while fighting is actually taking place. This will explain why the life of an army medical officer has been described as consisting of spells of intense activity alternating with periods of utter boredom. It also explains why a medical population that is equal to the needs of a community in peace-time, finds itself in difficulties as soon as the community goes to war. This is the present position in Australia. Early in the present conflict the difficulties were recognized and in order to deal with them a Central Coordination Committee was set up with subsidiary committees in every State. The story of these committees has been told in these pages on previous occasions. In some States useful action was taken, but in others dissatisfaction arose. Readers will remember that at the last meeting of the Federal Council of the British Medical Association in Australia it was announced that deputy chairmen were to be appointed to the Central Coordination Committee and to each of the State committees, that the deputy chairmen would be men who were in close touch with the practising members of the profession, and that more effective action might be expected in the future. The several committees had scarcely started to function under the new arrangements, and Major-General F. A. Maguire had only just taken up his new position

as Director-General of Medical Services and Chairman of the Central Coordination Committee, when a request came from Great Britain which demanded instant attention and action. Dr. J. G. Hunter, General Secretary of the Federal Council, received a cable from Dr. G. C. Anderson, Secretary of the Central Medical War Committee in Great Britain, asking whether Australia could send medical officers for service in Great Britain and India. Dr. Hunter immediately communicated with Major-General Maguire, who obtained ministerial approval to call a meeting of the deputy chairmen of the Central and State Coordination Committees and of the Deputy Directors of Medical Services of the several States. The meeting was held at Melbourne on April 19, 1941, and the whole position of the medical profession in Australia in regard to the war effort was considered. As a result of this meeting the Central Coordination Committee was called together on April 23. Major-General Maguire was in the chair and there were present: Surgeon-Captain Carr, Air Commodore Victor Hurley, Sir Alan Newton (Deputy Chairman), Sir Henry Newland, Dr. J. H. L. Cumpston, Dr. J. Newman Morris and Colonel Dowden of the Adjutant-General's department. After long and careful consideration the committee was reluctantly compelled to come to the conclusion that no Australian medical men would be available for service in Great Britain or India, as the Home authorities had wished. Major-General Maguire announced this decision at a large and enthusiastic meeting of the New South Wales Branch of the British Medical Association on April 24. He also addressed members on the whole question of the medical profession in Australia in relation to the war effort, and declared that he was making no reservations, since he wished the medical profession to see the position as he himself and the other members of the Central Coordination Committee saw it. Medical practitioners throughout the Commonwealth are looking for information; they want to know what is likely to be required of them; many, indeed, are anxious to serve, but so far have found no outlet to their energies; we therefore welcome the opportunity of putting the following facts before them.

There are in Australia something like 7,000 medical practitioners. The exact number is not known. The General Secretary of the Federal Council thinks that the number may be in the neighbourhood of 6,500 and possibly nearer to 6,000. In the following figures given by Major-General Maguire, however, the total of 7,000 has been taken as a starting point. Of the 7,000 medical practitioners 500 are women; some of these have married and have families or other home responsibilities. It is possible that even some of these might practise again and be of use in institutional services. Further, among the 7,000 there would be 1,170 males over sixty years of age, something like 250 would be medically unfit, and 500 would be engaged in essential services. When the four groups are taken from the pool of 7,000 there are 4,580 left. For the Royal Australian Navy, in addition to the medical officers at present serving, 15 will be required as reinforcements before the end of the present year; for the second Australian Imperial Force, in addition to the medical officers at present serving, 180 will be needed as reinforcements before the end of this year; for the Royal Australian Air Force 91 reinforcements will be needed this year. The three services, therefore, take 1,043 from the pool, leaving

3,537 males under sixty years of age. If mobilization should become necessary in Australia (and everyone will agree that provision must be made for this possibility) seven divisions in the field with corps and army troops would require field ambulances, casualty clearing stations and general hospitals with a medical personnel of 1,260, leaving approximately 2,280 medical practitioners to provide for a civil population of seven millions—a proportion of 1 to 3,100. Major-General Maguire points out that the position is not quite so serious as these figures would suggest, for many of the women doctors would be available, some of those males who are over sixty years of age would be able to do useful work, as would also some of those who are medically unfit for active service in the field. The present distribution of medical practitioners in relation to the population varies in the several States, and there is no need to reproduce the available figures. At the same time it must be pointed out that the position is somewhat complicated by the number of "one-man towns" and "two-man towns" in the Commonwealth. For example, there are in New South Wales 125 towns attended by one medical practitioner and 49 attended by two practitioners; there is no reason to suppose that the figures for other States would differ widely from these.

The present position in regard to men offering for service of one kind and another, as revealed by Major-General Maguire, is not satisfactory. Of recent graduates with one year's hospital experience only 112 have offered their services; 300 older men suitable for duties in hospitals have offered and 346 specialists. In other words, there are 758 men from whom it would appear that the 286 reinforcements required for 1941 can be drawn. But men for reinforcement duty should be of the younger graduate group, and it is for men of this class that the present need is most urgent. There is also urgent need for men to act as medical officers in militia camps.

Before the steps that will be taken to meet the present needs are discussed, attention must be drawn to some important points discussed by Major-General Maguire. He stated quite bluntly that the present difficulties were inherent in the voluntary system. With this most medical men and women will agree, for repeatedly the question of the conscription of the profession has been advanced as not only desirable but necessary. While it is unlikely that any Federal government would be willing to conscript one section of the community, it is an indication of the present state of medical preparedness that Major-General Maguire thinks that the time may come when the Government will have to be told that a state of emergency exists in relation to the medical profession. At present, however, this is rather outside the immediate needs of the situation. The authorities have shown wisdom in that they have given Major-General Maguire permission to employ medical men over military age for any service in Australia of which they are deemed capable. Heretofore medical officers for the second Australian Imperial Force have been trained in the units in which they left Australian shores. At present no medical units are in training for the Australian Imperial Force, and medical men destined to leave the Commonwealth as reinforcements will therefore be posted for home service that they may receive some training before they proceed overseas. Some training

is necessary before embarkation, and the camps are the only places where this can be done. Special courses have been arranged for medical officers in tropical medicine, in hygiene and in war medicine and surgery. Attendance at these courses, as Major-General Maguire pointed out, will be possible only if sufficient medical officers are available to permit a roster of leave from ordinary camp duties to be arranged.

Men willing to serve, particularly those who have recently graduated and have had hospital experience, are urged to indicate without delay their willingness to do so and to state whether they will be available at once or in one, three or six months. They are asked to communicate with the Deputy Director of Medical Services in the State in which they reside, or with the State Coordination Committee. The State Coordination Committees are to come prominently into the enlistment picture, for the medical heads of the three services will apply to these committees when they need men and will not choose men unless they have been approved by the Coordination Committee as suitable. Too much emphasis cannot be laid on the urgency of the need for younger men. The four Australian universities with medical schools are to be asked to shorten the medical course so as to speed up the graduation of students. Medical officers must be obtained for militia camps, and they will be obtained. Only 40% of the younger graduates have registered under the provisions of the Act which makes every man between the ages of 18 and 33 liable for service within the Commonwealth. It may be that some have not registered in the belief that medicine is a reserved occupation. It is a reserved occupation as far as service in the combatant ranks is concerned, but not in relation to service as a medical officer. Men who have not registered will be required to do so, and the vacant medical officerships in the home service ranks will be filled.

What has been written above covers most of the decisions recently made by the Central Coordination Committee. Medical practitioners who wish to have further information are invited to apply to the Deputy Director of Medical Services or to the Deputy Chairman of the Coordination Committee of their own State.

## Current Comment.

### GOUT, THE FORGOTTEN DISEASE.

GOUT is out of fashion. Most of us do not diagnose it. The fault, however, lies with us and not with our patients, if the recent claims of P. S. Hench are true. He states that about 5% to 8% of all patients in a joint clinic are suffering from gout. Interest in the subject has been scant for some years, but is now reawakening. In April last year C. G. Lambie reviewed in this journal certain aspects of gout and reported an exceedingly interesting case. John H. Musser<sup>1</sup> has recently contributed an extensive survey of the recent literature on the disorder, and the following is a short account of its chief features.

The disease has its onset most commonly during middle age, and almost exclusively in men. Women constitute only about 3% of the total sufferers. The disease is very rare in children, but is not unknown, an example having been reported even in a baby still at the breast. The cause is not well understood. There seems no doubt that heredity plays a part. The family incidence is high, and

<sup>1</sup> *The American Journal of the Medical Sciences*, January, 1941.

relatives of affected persons have been found to have an increase of uric acid in their blood, though free of symptoms. The uric acid in the blood of the patient almost always rises before an attack, recent work suggesting that this is due to increased formation in the body. It seems likely that some disorder of the liver is associated with this rise. The liver plays an important role in purine metabolism and is enlarged in many gouty patients. A considerable amount of work has been done in an attempt to find what relationship exists between the kidney and this disease, but little except interesting observations have as yet resulted. The first of these is that a marked diuresis occurs during the one or two days before an attack and continues while the attack is at its height. The second is that though chronic renal impairment is the rule in gout, it is not invariable, and it therefore seems unlikely, as has been suggested, that a primary renal disease causes the rise in the blood uric acid content by hindering excretion and so the characteristic symptoms. It has been suggested too that the disease is allergic in nature. The most impressive evidence in favour of this suggestion is not that some patients have other allergic manifestations as well as gout, but that the acute attack of gout frequently follows a dietary indiscretion of a certain type. This, however, is poor evidence, for there are many other factors that can precipitate an attack. Trauma, operations on parts remote from the joints, alcohol, foods rich in purines and certain drugs, particularly gold, "Salyrgan", lead, thiamin and ergotamine have been known to play the same role. Of considerable interest are the cases reported of attacks occurring in anaemic patients who are showing a rapid erythropoietic response to haematinics. It is suggested that the breakdown of the nuclei of the many red blood cells maturing in the bone marrow is the source of the purines that precipitate the attack.

It would seem fortunate, then, that gouty patients are rarely anaemic. The blood count seldom shows any deficiency in red cells or haemoglobin, and so the florid complexion that picks out the sufferer from gout among the other habitués of a clinic for chronic joint diseases, among whom anaemia is so common. Characteristically gout consists of a series of acute attacks at variable but often long intervals. Between these attacks the patient is completely free from symptoms, an important diagnostic point. The acute attacks most often have their onset at night and last for several days. The classical description of a uniarticular lesion in the great toe still holds good, particularly for the original attack. It is estimated that 60% of men patients have their first attack in this site. Such examples of the disease can hardly be diagnosed incorrectly. What recent writings stress, however, is that while these cases may be most common, their majority is not large. Almost any joint or several together may be attacked. Any patient with recurrent attacks of sudden acute arthritis and complete freedom from symptoms between attacks should be suspected of having gout, no matter what joints are affected. The feet, hands, knees, elbows, shoulders and hips have been repeatedly involved. If the fingers are involved the distinction from acute rheumatoid arthritis may be difficult. More perplexing is the group of patients who have reached the stage of suffering from chronic joint pains. If these tell that their illness was early characterized by isolated attacks, the diagnosis may still be gout, for chronic gout not infrequently follows a number of acute attacks. It should be remembered too that when the disease occurs in women it tends to be atypical. In a proportion of patients, variously estimated at from 25% to 58%, tophi develop and enable the diagnosis to be established beyond doubt. They may occur on the ear, elbow, eyelid, conjunctiva, fingers or toes. They appear first as small red swellings, which tingle and burn. They may break down and discharge. Not infrequently they give no sensations at all. They are firm in consistency and contain a collection of precipitated urate crystals.

Laboratory aids are not of great use in the diagnosis. The level of uric acid in the blood is almost but not quite invariably raised. Leucocytosis and raised blood

sedimentation rate are the rule during an acute attack. There is seldom anaemia. The urine is usually acid. It is not true, however, as has been stated, that an alkaline urine is incompatible with a diagnosis of gout. Radiological examination of the long bones may reveal eroded punched-out areas at or near the joints when the disease has reached its chronic stage. A number of less typical changes, of no real use in diagnosis, sometimes occur. During the stage of acute attacks no changes are seen by X-ray examination.

A final useful procedure to help establish the diagnosis of a difficult case is a therapeutic test with colchicum, which remains the most effective drug available, and should give relief in one or two days. Various authors give it as colchicine in doses of about one one-hundredth of a grain every one to three hours till pain is relieved or diarrhoea takes place. Cincophen may be used, but cases of poisoning from this drug continue to be reported. Recently thiamine chloride has been used in the chronic stage. It may provoke an acute attack, but shows promise of reducing the chronicity of the disease. Heat, rest and diet are the other long-established elements of accepted treatment.

Though there have been no recent outstanding developments in the study of gout, we call attention to Musser's review. Gout is a disease that is still among us, and it must not be forgotten. It should automatically figure in every physician's differential diagnosis of acute or chronic joint disease, not because it is common, but because it most assuredly will not be diagnosed as often as it should be unless it is constantly thought of as a possibility.

#### THE IODINE CONTENT OF THE BLOOD.

The iodine content of the whole human adult is small; it varies between 20 and 50 milligrammes. Of this quantity it is said that the muscles contain one-half, the skin one-tenth and the skeleton one-seventeenth. The total iodine in the blood contributes less than one-tenth of the whole iodine in the body—that is, less than two to five milligrammes. It will therefore be realized that the estimation of the iodine in a small quantity of blood is a very difficult and delicate chemical procedure and demands considerable analytical skill. It has been estimated at less than 10 microgrammes per 100 cubic centimetres of blood, but according to William Salter,<sup>1</sup> it is possible for several microchemists to analyse the same sample of blood and report values for iodine ranging from 7 to 70 microgrammes *per centum*. Salter's comment that technique is still a major problem is quite a justifiable one. Isolated values obtained by different procedures in various localities are of little use; comparative values obtained by the same investigators are more reliable. Obviously every worker must establish his own range of normal values.

Technique, then, is the initial difficulty, but not the only one. The total blood iodine concentration at a given moment represents the net result of many complicated processes. Vigorous exercise causes an increase in the fasting blood iodine within a few minutes. Increases of over 150 microgrammes *per centum* have been recorded. Mental excitement increases iodine elimination. Variations in body temperature affect it. The effect of the administration of iodine is interesting, for iodides are absorbed very rapidly, and after doses of two grammes or more the concentration of blood iodine may increase by more than 1,000 microgrammes *per centum*. The effects of administration of thyroid derivatives have not been well studied. In regard to thyrotoxicosis, it is generally stated that the blood iodine level does not run closely parallel to the severity of the disease. Studies of the iodine content of the blood in thyrotoxicosis are being made by various investigators, notably in Boston; but it is obvious that the time has not yet come when such studies can be of use to the clinician. Statements in text-books, claiming that the estimation of the blood iodine content is of value as a diagnostic aid, must be regarded, at present, with scepticism.

<sup>1</sup> *Physiological Reviews*, July, 1940.

## Abstracts from Medical Literature.

### RADIOLOGY.

#### Calcified Lesions in the Spleen.

H. C. SWEANY (*American Journal of Roentgenology*, August, 1940) states that in a study of a large series of cases the evidence has been practically all in favour of tuberculosis as the cause of calcification in the spleen. First, calcification is within the spleen parenchyma and not in the splenic veins outside the spleen or in the trabecula, where phleboliths are wont to appear. Most calcification occurs in multiple areas and seems to result from haemogenous dissemination. Many of the areas in each group of calcification are two or three times the size of the largest veins, and therefore too large to be phleboliths. They are all associated with and correspond roughly in age characteristics to a primary complex elsewhere (in the neck, lung or mesentery). Therefore, they would probably correspond to the haemogenous phase of the primary infection. They occur in tuberculous individuals, most of whom show a continuous progression of tuberculous disease; moreover, the great majority of these persons are free from pathological and serological evidence of syphilis or concrete evidence of other interfering diseases. When examined microscopically these areas have no resemblance to phleboliths; the vascular walls of smooth muscle may be seen, and the internal structure is different from tubercles. The finding of calcified tubercles in the spleen has little clinical significance *per se*, except to show that primary tuberculosis with haemogenous dissemination once existed and to help to exclude other similar densities in the splenic region.

#### On Tuberculosis of Bronchial Lymph Glands.

NILS WESTERMARK (*Acta Radiologica*, August 30 and October 31, 1940) states that when enlarged hilar lymph glands are present it is a difficult problem to decide whether this enlargement is the result of tuberculous changes or is due to some other pathological condition in the gland. A bronchopneumonia even of slight extent may cause a more or less pronounced hilar lymphadenitis. In these cases, too, the lymphadenitis is regional to the pulmonary field. A hilar lymphadenitis in connexion with bronchopneumonia is rarely, however, of the same extent and density as tuberculous lymphadenitis. At the first examination it may be impossible, however, to make a definite radiological differential diagnosis. The bronchopneumonic changes as well as the consequent hilar lymphadenitis run a very acute course, the changes generally disappearing fairly soon or diminishing considerably. On the other hand, it is found that a primary tuberculous focus with consequent hilar lymphadenitis runs a more chronic and often progressive course. A tuberculous primary focus and tuberculosis of hilar lymph glands clear up very much more slowly than bronchopneumonia with secondary lymphadenitis. In the differential diagnosis between tuberculosis of hilar lymph glands and other forms of hilar lymphadenitis bronchography is frequently of great use. At the seat of the primary tuberculous focus multiple, evenly rounded, stenotic alterations of

the bronchial branches are found. These stenotic conditions are caused by a purulent exudate which fills up and infiltrates the bronchial lumina. In ordinary bronchopneumonia, bronchographic examination reveals no radiological signs of changes in the bronchi.

#### The X-Ray Aspects of Non-Putrid Pulmonary Suppuration.

MARCY L. SUSSMAN (*American Journal of Roentgenology*, September, 1940) states that non-putrid pulmonary suppuration is a severe form of bronchopneumonia in which necrosis of pulmonary tissue takes place. Until an abscess can be demonstrated, the diagnosis of suppuration rather than simple bronchopneumonia cannot be made by X-ray examination. Areas of rarefaction seen within a pneumonic shadow may represent foci of suppuration, but differentiation from a resolving pneumonia is impossible from the single film. However, the diagnosis should be considered when these areas persist, become larger or become confluent. The diagnosis of abscess can be made only when a fluid level is demonstrated. Cavitation within several areas of a diffuse suppurative bronchopneumonia may be an incidental radiological finding and may have little effect on the clinical course of the disease. It does not necessarily take place in all of the pneumonic areas at the same time. In fact, it is more likely that the pneumonic areas will be found to be at different stages of formation or resolution. While it is probable that, in most cases, an area of rarefaction outlined by a fluid level is an abscess, there is the possibility that in some cases an infected emphysematous bleb is present. When more than one fluid level is present the X-ray examination does not permit the differentiation of solitary multilocular abscess and multiple abscesses. Although non-putrid abscess tends in general to spontaneous resolution, occasionally, in the presence of a clinical cure, a cavity is found to persist for many years or perhaps permanently. The appearance is that of a cyst-like structure within the lung, with or without a thin rim of infiltration. Persistent cavities may follow both putrid and non-putrid processes. They often remain asymptomatic, but occasionally are the source of further lung disease. Non-putrid pulmonary suppuration is often presented to the radiologist when pleural complications have already occurred, obscuring the underlying pulmonary lesion. Since purulent bronchitis with destruction of the elastic tissue of the bronchioles is a constant feature of suppurative bronchopneumonia, bronchial dilatation occurs regularly, often within a few weeks. Mild bronchial dilatation is not necessarily permanent. In many of the cases studied restoration to normal occurred within one year.

#### The Grooved Defect of the Humeral Head.

HAROLD A. HILL and MAURICE D. SACHS (*Radiology*, December, 1940) state that compression fractures as a result of impingement of the weakest portion of the humeral head, that is, the postero-lateral aspect of the articular surface, against the anterior rim of the glenoid fossa are found so frequently in cases of habitual dislocation that they have been described as the "typical defect". If large, such grooved defects may lead to unsatisfactory results following the usual operations for recurrent dislocations. A minimal X-ray examination

comprises an antero-posterior projection of the shoulder with the arm in adduction and external rotation, and a similar projection with the arm in marked internal rotation. Tangential views often prove useful. The X-ray characteristics of posterior humeral compression fractures are: a wedge-shaped defect of the postero-lateral aspect of the head of the humerus, varying from five millimetres to three centimetres in length, three millimetres to two centimetres in width, and 10 to 22 millimetres in depth. A special sign is the sharp, vertical, dense, medial border of the groove known as the "line of condensation", the length of which is correlated with the size of the defect.

#### Insufficiency Fracture of the Tibia Resembling Osteogenic Sarcoma.

GEORGE E. PFAHLER (*American Journal of Roentgenology*, February, 1941) states that insufficiency fracture is an incomplete fracture of the cortex of the bone, resulting from excessive strain to which the bone has not yet become accustomed, such as occurs in military service in new recruits, or in the strenuous physical exercises of gymnastic work or athletics. It affects one side of the bone. It results from single or from multiple strains. In the majority of cases that have been reported, no fracture line could be detected on X-ray examination in the early stage. It seems to develop later, after a month or more. The first symptom is pain on exertion, localized at the site of the lesion. This is followed by swelling of the periosteum of the bone, or rather the appearance of callus formation. The early swelling felt over the painful region is thought to be due to an edematous condition. These relatively uncommon cases of insufficiency fracture occurring in the large long bones are similar to those which occur commonly in the second, third and fourth metatarsal bones and which have been especially described under the term "march fracture". The author reports a case in which the tibia was involved, and issues a warning of the danger of making an erroneous diagnosis of sarcoma. The fracture lines are frequently so fine that they are difficult to detect even with the use of a magnifying glass.

#### Multiple Cystic Tuberculosis of Bones.

JOHN L. LAW (*Radiology*, September, 1940) describes the X-ray appearance of multiple cystic bone tuberculosis and illustrates the healing process by a series of skiagrams taken in a case characterized by widely disseminated lesions. The condition is a comparatively rare form of diaphyseal bone tuberculosis, usually multiple, and of metastatic origin from a primary thoracic focus. It is insidious in onset, with few or no localizing signs, such as pain, tenderness or external evidence of inflammation. The lesions run a chronic benign course and consist of a circumscribed area of soft tuberculous granulation tissue and degenerated bone, which tend to heal slowly. Two main radiological types are described: the diffuse and the circumscribed. The diffuse is the more acute initial stage. In it the diaphysis cannot be differentiated into medulla and cortex. Here the cystic areas of decreased density are honeycombed with a web-like structure of increased density. The individual or several apertures in the web vary in size from that of a millet seed to that of a pea. The cystic areas may

enlarge to include both medulla and cortex, increase the diameter of the involved bone, and cause destruction of a phalanx. In the circumscribed type the skiagram shows round smooth or irregular areas of decreased density, with a punched-out appearance and a thin cortex and little or no surrounding sclerosis. Usually no involvement of the periosteum, no cortical expansion and no abscess or sequestrum formation are present.

#### Variations in Calcification Pattern in the Epiphyses.

L. W. SONTAG AND S. L. PYLE (*American Journal of Roentgenology*, January, 1941) state that a period of very rapid growth, particularly in the broader epiphyses of the skeleton, often produces a ragged appearance. Calcified areas may appear completely detached from the bony nuclei. A greater or lesser degree of the appearance is an almost constant finding in the distal femoral epiphysis, and in a mild degree it is not infrequent in the proximal tibial epiphysis. The ragged appearance is due to a period of very rapid epiphyseal growth, which is usually a part of very rapid general skeletal growth. It is entirely a maturational and growth factor. The age at which the process occurs and the degree of its floridity are influenced by thyroid function and changes in thyroid function.

#### PHYSICAL THERAPY.

##### Röntgen Irradiation in the Treatment of Inflammations.

E. P. PENDERGRASS AND P. J. HODGES (*American Journal of Roentgenology*, January, 1941) state that there is still much scepticism as to the value of irradiation in the treatment of inflammations, largely owing to the lack of well-controlled experimental evidence supporting this use of irradiation. Opinions concerning the mechanisms by which irradiation influences inflammation vary considerably. This may be accounted for by the lack of reliable information concerning the exact nature of the biological processes enacted after irradiation and the confusion which still exists in the field of immunology. The authors discuss these matters at length. Although radiation in huge quantities is bactericidal, there is no evidence in the literature that the small doses of radiation commonly used for infections have this action. Desjardins and others believe that the destruction of leucocytes by irradiation sets free protective antibodies more quickly than they are usually set free in the natural course of inflammatory processes. It is possible that this action is of more significance in the treatment of chronic than acute infections. Another theory, supported to some extent by experimental evidence, is that irradiation alters the albumin-globulin ratio in the blood and so enhances antibody formation. Dilatation of the capillary network and subcapillary plexus is a uniformly characteristic response to small doses of radiation (200 to 300 r). This active hyperaemia is thought to be of considerable importance in the treatment of infections. The results of the treatment of various types of infections by X rays are analysed. In bursitis approximately 60% of lesions showed rapid improvement after irradiation. True carbuncles, when

seen early, respond readily to irradiation and may be aborted. In cellulitis, 78% of lesions showed rapid improvement after X-ray therapy. The writers have had little experience in the treatment of cellulitis or lymphangitis with sulphamamide plus irradiation. In erysipelas, the combination of X rays and sulphamamide has not been found harmful. Of 36 patients with this disease, 30 showed rapid improvement in one or two days. The value of irradiation in pyogenic granuloma, *herpes simplex*, acute parotitis, pneumonia and sinusitis is discussed.

#### The Effect of Röntgen Irradiation on the Lymphatic Transport of India Ink.

E. D. SUGARBAKER AND K. SUGIURA (*American Journal of Roentgenology*, November, 1940) have attempted to determine experimentally whether or not irradiation facilitates the immediate lymphatic dissemination of cells. For many years the objection has occasionally been raised that irradiation appears to be responsible in some cases for the widespread dissemination of cancer cells. It is undoubtedly true that occasionally cases occur in which rapidly advancing metastases develop either during or shortly after the course of treatment. The authors have conducted experiments in which India ink diluted with water was injected into the pectoral region of young rats, and then twenty-four hours later the animals were irradiated. A control group of animals was injected with the ink, but received no irradiation. At a later date the animals were killed and careful examination of the axillary and mediastinal glands was made. The results showed that a lesser quantity of pigment was present in the glands of the irradiated animals as compared with those of the controls. This suggests that neither injury to the lymphatic vessels nor increase in lymph flow occurs as a result of the irradiation. It therefore seems probable that metastases becoming clinically apparent during the course of radiation treatment were already present in subclinical form before treatment was begun.

#### Radio Interference Problems from Operation of Electro-Medical Apparatus.

E. K. JETT (*Archives of Physical Therapy*, November, 1940) points out that interference is the problem child of radio and one of the principal reasons for government regulation of it. In 1935, in the United States of America, commercial and government wireless stations began to experience interference on the high frequencies used for long-distance communication. An investigation finally disclosed that the interference was caused by diathermy machines operated by private doctors and in medical centres. The reason that the operation of diathermy apparatus affects radio reception is because the machines are essentially radio transmitters. Radiations from these machines could be controlled by the ordinary telegraphist's key and are capable of being received across the continent and even across the ocean. The frequencies, too, on which they operate are those used by the national services. The interference can, however, be eliminated or immunized if reasonable methods are employed. Screening of the apparatus or room is the common method. When screening is not practicable or advisable, it would

appear that the only solution would be to use apparatus with a restricted frequency band of omission which does not extend over more than one communication channel. A decision has first to be made as to the most effective region of the radio spectrum for diathermy treatment; and then the task is to find a suitable frequency for diathermy operation.

#### The Optimum Dosage in the Treatment of Carcinoma of the Uterine Cervix by Radiation.

M. C. TOD (*British Journal of Radiology*, January, 1941), in order to assess the dose of radiation therapy actually delivered in 370 cases of carcinoma of the *cervix uteri*, has used the method of assessing the dosage in Röntgens in the paracervical tissue which has been developed for this purpose at the Holt Radium Institute, Manchester. It had been shown that the limit of tolerance to treatment by radium placed in the cavity of the uterus was not determined by the dose on the vaginal surface or at the external os, but by the dose to the paracervical tissue; and a point A was selected two centimetres lateral to the uterine canal and two centimetres from the mucosa of the lateral fornix as an index of the dose in this region. The net survival at three years after treatment, the dose at point A in each case, and the total time taken to deliver the dose are considered together. Only one patient in the series with a dose of less than 6,000 r at point A survived for three years. It should be realized that a dose of less than 6,000 r may be obtained at A even with high dosage at the external os or vaginal surface. Pending further study with predetermined dosage, the following would seem to be a fair statement of optimum dose levels at point A: (i) 7,500 r to 8,500 r in six to seven days when radium only is used; (ii) 8,500 r to 9,500 r for three or four serial weekly applications when radium only is used; (iii) 9,500 r to 10,500 r summated dose for combined radium and deep X-ray treatment over four to six weeks.

#### Treatment of Advanced and Recurrent Carcinoma of the Breast.

W. S. PECK, H. K. RANSOM AND F. J. HODGES (*American Journal of Roentgenology*, December, 1940) review the case records of 920 patients with breast carcinoma admitted to their hospital during the period 1931 to 1938. More than half of these either had inoperable growths on admission or were subsequently shown to have recurrent or metastatic lesions. The authors have no doubt that irradiation is the most effective single agent in the treatment of advanced and recurrent breast cancer. In certain cases mastectomy following irradiation will frequently improve the palliative result. Recurrences were found to be rare in the older patients with slowly growing carcinoma treated by mastectomy and irradiation. Castration by irradiation is recommended when remote metastases or local recurrences have appeared and if the patient is menstruating. Treatment, however, must be decided in each individual case, and the castration of all patients who have had breast cancer does not seem justified. Routine treatment of all demonstrable metastases is not recommended; but lesions producing symptoms or causing disintegration of the weight-bearing skeletal system should be treated by irradiation.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on March 5, 1941, at the Medical Society Hall, East Melbourne. Dr. H. BOYD GRAHAM, the Acting President, in the chair.

#### Modern Aspects of Puerperal Sepsis.

DR. A. M. HILL read a paper entitled "Modern Aspects of Puerperal Sepsis" (see page 537).

PROFESSOR MARSHALL ALLAN congratulated Dr. Hill on his masterly survey of modern views on puerperal sepsis. Much of the advance which had been noted was due to the bacteriological research work of Colebrook and to the report of Smith of Aberdeen on the importance of droplet infection. Dr. Hill had stressed the modern classification, particularly of the streptococcal group of organisms, and it was important to note that in Melbourne at least the anaerobic organisms were a greater source of infection than those of group A streptococci. There was a tendency in many reports to consider that the latter group alone was of importance. A knowledge of the changes in morbid anatomy had been made much clearer when they were discussed in respect of each infecting organism.

Emphasis on the principles of treatment was still necessary, and in particular the need for efficient masking. From a knowledge of recent outbreaks of sepsis Professor Allan had been astonished to find that masking had not been universal and that in some instances attendants with nasopharyngeal disorders had been allowed to remain on duty. There was a legal responsibility in the event of an action at law, which should not be overlooked by all concerned.

A recent article in *The British Medical Journal* on surgical infections in war wounds was very timely. While the surgeon tried to guard carefully his patients in the operating theatre, a certain degree of laxity often crept into the after-care service. The use of sulphanilamide would solve only some of the problems, and it was still necessary for medical men to educate attendants and to watch themselves for any possible avenue of infection. That meant constant watchfulness based on knowledge and intelligence.

Professor Allan said that three years earlier he had stressed in a circular issued by the Department of Health the need for the provision of a speedy, uniform and cheap bacteriological service available to all practitioners, as well as the establishment of a central isolation block closely associated with the Women's Hospital and available for all grades of patients. Neither of those fundamental requirements was yet available. In conclusion, Professor Allan remarked that though many problems still awaited elucidation, Dr. Hill had shown clearly how to diagnose and treat puerperal conditions as they arose, and his paper was a valuable guide from one who had an international reputation in such matters.

DR. A. M. WILSON said that he always found Dr. Hill interesting; but he would go home that night with cold shivers down his back. The only hope in puerperal sepsis was in its prevention; one could do much good by attention to detail. He was glad that Dr. Hill had stressed the importance of reducing to vanishing point vaginal examinations during the confinement and puerperium. A rare combination of scientific knowledge and "horse-sense" was required in the successful conduct of labour. It was necessary to steer between the courses of expectant reliance on nature and exhaustion of the patient; it was wise to concentrate attention not on what she could endure, but on what she could accomplish.

Dr. Wilson said that he had little to add; but he wished to ask Dr. Hill to express an opinion about the use of sulphydryl in prophylaxis in a case of "failed forceps" delivery. Dr. Wilson was in the habit of advising against it, because the patient would be very ill and might be made to feel very nauseated. He relied on a combination of rest, sleep, food, and a preparation rich in vitamin A, such as "Radiostoleum" or "Adexolin". Dr. Wilson had felt satisfied with those measures and had given up the use of anti-streptococcal serum. All obstetricians at times ran into trouble with forceps and were sometimes in a quandary as to what to do for the best.

Dr. Wilson then observed that the excellent treatment outlined by Dr. Hill cost a great deal of money, and while the very rich could get it by paying for it and it was available for the poor at such places as the Women's Hospital, it was extremely difficult for the large group of patients who came in between these two groups. He strongly

advocated the provision at the Women's Hospital of a large intermediate block for the confinement of women of the middle class. He added that it was extremely difficult to find enough maternity beds for them in Melbourne, and that recently with the arrival of so many young brides from abroad the situation had become intolerable; indeed, he had just been successful in obtaining a bed for a patient who was due to be confined on October 10, after many hospitals had regretted the inability to accept the booking.

DR. B. M. SUTHERLAND congratulated Dr. Hill especially on the way he had condensed his material; he had completely covered in one hour the ground that would have occupied three addresses in routine lecturing, and yet no one could make any significant additions.

With reference to the prevention of sepsis, Dr. Sutherland referred to the avoidance of certain predisposing factors, such as the subjecting of the patient at confinement to exposure to the coldness and dampness associated with lying on a sheet with rubber sheeting beneath it from the time of the rupture of the membranes until the arrival of the baby. Particularly when the patient had had a basal anaesthetic, the administration of nourishment was apt to be left out; and excessive loss of blood in the third stage occurred at times, even when partial separation of the placenta could be diagnosed by the gushing of blood with each contraction. Dr. Sutherland added that the manual removal of the placenta at an early stage was indicated in partial separation and was a safe procedure if careful surgical technique was followed.

Dr. Sutherland then went on to emphasize the importance of wearing masks to prevent droplet infection, urging that the medical practitioner should wear a mask and insist that the nurses present should do so too. He had heard that a legal decision had been given in the United Kingdom awarding damages for malpractice on the grounds of refusal to wear a mask, and he thought that damages for puerperal sepsis might follow proof of failure to wear a mask, much in the same way as in fracture it was regarded as negligence if no X-rays were taken. In conclusion, Dr. Sutherland stressed the early onset of peritonitis in the puerperium; he mentioned the transient pain on the second day which led to the diagnosis of the aplastic type of rapidly developing peritonitis. He said that that was the stage for surgical interference and that drainage at a much later period was practically valueless.

DR. W. R. D. GRIFFITHS spoke of the value of the use of preparations containing vitamin A (such as "Radiostoleum" and "Adexolin"), which seemed to influence the rise and fall of the temperature. He thought that as a rule anti-streptococcal serum was valueless; but if the sepsis was associated with a scarlatiniform rash, the special scarlet fever antitoxic serum could act like a charm, placing the patient out of danger within thirty-six hours.

DR. R. G. WORCESTER referred to a case of septicaemia from *Staphylococcus aureus* in which the patient contracted pneumonia. He said that it was difficult to decide whether a transfusion of blood should be given to a patient with pneumonia. He said that he could support the contention that it was difficult to obtain accommodation for midwifery patients; he had one due to be confined on October 1 whom he had not yet been able to place. He was in agreement with Dr. Hill's remarks concerning masking, particularly during the puerperium, and quoted an instance of a nursing sister who had been in attendance on a patient in the puerperium while suffering from an acute antral infection.

DR. F. V. SCHOLES said that he had come to the meeting to pick up crumbs by the wayside. It was news to him to learn of the high morbidity and mortality rates for which the anaerobic streptococci were responsible, and he considered Dr. Hill's classification masterly. Dr. Hill had tabulated the lesions of the morbid anatomy of the processes and had described the different methods of spread and the lesions produced by the organisms under discussion. Dr. Scholes assumed that sulphydryl was the drug of choice, because at first it was difficult to know which germ was involved; it should be continued if the pneumococcus was incriminated, but "Proseptasine" was to be preferred against haemolytic streptococci. He went on to say that the importance of obtaining an effective concentration of the preparation used at an early stage was generally recognized, but mentioned that he had himself gone far beyond the usual dosage and had used 20 grammes for an adult within a few hours. At the Queen's Memorial Infectious Diseases Hospital they saw some examples of puerperal sepsis and septicaemia; he remembered a group of seven of them in the past year. All the patients had been infected at one midwifery hospital and had had the scarlet rash and other features. In a typical case at the Infectious Diseases Hospital, when a patient came under treatment perhaps on the third day, with a high temperature, scarlet fever

antitoxic serum would be given, together with "Proseptasine" or sulphapyridine; the temperature would probably be normal within twenty-four hours and the patient would feel well within forty-eight hours. Dr. Scholes put in a plea for the use of scarlet fever antitoxic serum in infection with haemolytic streptococci of the A group associated with high fever.

Referring to the question of masks, Dr. Scholes said that he had discussed elsewhere the advisability of wearing masks in routine fashion in open wards in ordinary medical cases. At least the practice would have the moral value of making the wearer take more care to avoid the spread of infection; the human hand was the most potent instrument, and the next was close droplet infection from direct coughing. Droplet infection had been studied for forty years or more from that point of view, though it was true that only for a few years had attention been focused on its importance in puerperal sepsis.

Dr. J. S. GREEN described a dramatic but unhappy experience in puerperal sepsis. Though the confinement had been conducted without undue handling of the patient, she soon became feverish and had a rash; scarlet fever antitoxic serum was used and was followed by a crisis, and the rash faded. The patient relapsed and improved several times, but died in three weeks. Dr. Green thought that the timely use of transfusion of blood was of prophylactic value. Manual removal of the placenta should not be embarked on light-heartedly; if haemorrhage was excessive, blood transfusion should not be delayed. Agranulocytosis from treatment was another risk which had to be guarded against and which might cause much worry.

Dr. Green then referred to an account of the occurrence of sepsis at the Rotunda Hospital, Dublin, between 1822 and 1832. When there was no interference with the patient, the mortality rate in some 25,000 cases was approximately 0·5%; the rate in forceps cases was only 24 in 16,000, though between 60 and 70 times the fetus was macerated or destroyed. Dr. Green added that the patient was in the ward only for a short time, that the walls were whitewashed daily, and that chloride of lime was spread on the floor. He then emphasized the importance of proper masking, and advocated the compulsory use of wall diagrams to show how to employ the mask in the approved manner. In conclusion, Dr. Green acknowledged the value of Dr. Hill's work and influence at the Women's Hospital.

DR. C. H. DICKSON said that as the question of legal liability had been raised by a previous speaker, he wished to direct attention to a legal case report, in which the plaintiff had obtained a verdict and substantial damages from a certain hospital, to which the admission of midwifery patients had continued in spite of an outbreak of scarlet fever. In a decision of the House of Lords, it was laid down that the managers of an institution must draw attention to such a peril. Dr. Dickson added that any practitioner concerned ran the risk of being joined in such an action.

DR. J. B. G. MUIR (North China) said that he had for many years been associated with the medical department of a large British industrial concern, with the entire charge of a large community, including a full maternity service. The average foreign patient engaged a private medical practitioner; the Chinese were conservative, and confinements were conducted in their small mud huts and only came under the notice of his department when complications arose. Chinese women were being urged to attend antenatal clinics and to enter hospital for confinement. Puerperal sepsis was not prevalent, but as they met it in the hospitals it was very severe and usually the result of neglect. Gas infection was quite common, and often the sepsis followed attempts to treat at a late stage such complications as an impacted fetus in a persistent occipito-posterior position. The patients suffering from sepsis were nursed in widely separated and completely isolated blocks. In their midwifery work destructive operations, such as perforation, decapitation and evisceration were at times necessary. Osteomalacia was common, especially around Peking. Miraculous results had been produced by the combination of the lower segment operation and massive dosage of drugs of the sulphanilamide type. Dr. Muir considered it a great mistake to attempt repair operations in the presence of the least sign of infection. He had used sulphanilamide in dosage amounting to as much as 10 grammes every four hours for two days, and had found that hot lemon drinks helped the patient to retain the drug. He added that he had never seen a patient recover from fulminating peritonitis, though it was possible sometimes to save those with localized peritonitis; drainage by suction, fluids given by the "drip" method, operation and sulphanilamide treatment were most satisfactory.

DR. JOHN GRAY emphasized the value of antenatal care in the prophylaxis of sepsis. The early recognition and timely treatment of complications minimized the difficulty at the actual confinement. Common preventable factors were such things as a shortage of sterile towels and sheets. Dr. Gray asked Dr. Hill to express an opinion on the efficacy of deep X-ray therapy for *Clostridium welchii* infections and to say something about the employment of nursing trainees with but little experience at confinements in midwifery hospitals.

DR. BOYD GRAHAM, before calling on Dr. Hill to reply, undertook to bring the problem of the shortage of midwifery beds to the notice of the members of the Council of the Victorian Branch of the British Medical Association at the earliest opportunity.

Dr. Hill, in reply to Dr. Wilson, said that he considered that the sulphonamide drugs should be given early if the patient became feverish; they had an antibacterial effect and were particularly beneficial while the bacteria present were relatively few in number. If the drug was discontinued, it could be used again when the indication to do so appeared. Dr. Hill remarked that the legal point raised by Dr. Sutherland was important. He informed Dr. Griffiths that there had been only one patient with puerperal scarlet fever at the Women's Hospital in three years, and that he had had no personal experience of the value of scarlet fever antitoxic serum in puerperal sepsis; however, the reports in the literature were favourable. There was no proved work showing the advantage of giving any serum; it was necessary to check the bacteriological findings in every case. "Proseptasine" had certain advantages; it was least toxic, easiest to take, best for children and best in mild sepsis. On the other hand, it was not best in severe cases, as it was not well absorbed, and a sufficiently high concentration in the blood could not be obtained. Sulphapyridine was the most potent drug of the group, and it was wiser to use it in established sepsis.

Dr. Hill agreed with what Dr. Muir had said about repair operations. It was wise to leave lacerations open until the danger of sepsis had passed; often when they were closed they had to be opened later. Both Dr. Muir and Dr. Scholes had mentioned large dosage of sulphonamide; if it could be taken, it would probably be an advantage to the patient. No organisms could compare with *Clostridium welchii* in the cataclysmic downward race; it was hard to arrange for deep X-ray treatment before the patients died. There was no justification whatever for nursing trainees to take over delivery in private cases, and indeed it was illegal. In conclusion, Dr. Hill thanked those who had contributed to the discussion and the members present for their kindly references to his work.

## Medical Societies.

### MELBOURNE PEDIATRIC SOCIETY.

A MEETING of the Melbourne Pediatric Society was held on March 12, 1941, at the Children's Hospital, Melbourne, Dr. H. L. STOKES, the President, in the chair.

#### Osteomyelitis near the Hip Joint.

DR. H. DOUGLAS STEPHENS showed a boy, aged six years, suffering from osteomyelitis near the left hip joint. Dr. Stephens showed in skigrams the site of the lesion in the great trochanter, observing that there was no evidence of thickening or involvement of the joint. As, however, the boy's sister had died comparatively recently from tuberculous meningitis, and as the boy himself reacted strongly to the Mantoux test and also to a tuberculin patch test, it was extremely difficult to exclude a tuberculous origin.

From November, 1939, for nine months, the boy had been under treatment in a double ambulatory knock-knee splint. By August, 1940, his legs were straight, and he walked well for three months, until in December he was seen to be limping with the left leg and to have slight wasting of the thigh, but no limitation of movement at the left hip joint. During the period of three months between examinations he lost eleven pounds in weight. On general examination some of the upper deep cervical lymph glands were found to be enlarged, but not tender, and a few "shotty" glands were palpable in the left inguinal region. Clinically, the condition of the heart, lungs and abdomen was satisfactory, and no abnormalities were observed in the skigrams of the chest. In those of the pelvis the lesion was seen to be situated in the neck of the left femur up to the epiphyseal

line. The radiologist's opinion was that the appearances were in favour of a tuberculous rather than a coccal lesion and that the joint space was not narrowed. The movements at the left hip joint were good and there was no pain; a slight degree of limitation of extension appeared to be present, and a little tenderness was elicited on firm pressure over the great trochanter.

Dr. Stephens said that there had been delay in active treatment, because diphtheria organisms had been cultured from a routine swabbing of the throat. He had contemplated operation on the bone, on the assumption that it contained a localized low-grade pyogenic lesion with sequestration which had not yet extended into the joint; he thought that it was inevitable that the joint would be involved if conservative treatment was followed. He was hesitant to operate, however, because he was unable with certainty to exclude the tuberculous factor.

DR. W. KENT HUGHES thought that the aetiological diagnosis could be established at the exploratory operation and that no harm would be done if the lesion was tuberculous.

DR. W. R. FORSTER gave some details of a somewhat similar case that he had taken over from Dr. D. O. Brown at the Frankston orthopaedic branch of the hospital. The radiographic appearances were very similar and the joint was not affected, but complete healing had taken place within one year. In the light of that experience Dr. Forster did not think that Dr. Stephens should operate on the bone; one of the lymphatic glands in the iliac group might, however, be removed for biopsy to help to arrive at an accurate diagnosis.

DR. J. G. WHITAKER expressed the opinion that the lesion was tuberculous; but as there was no synovial involvement at the present stage, definite signs and symptoms had not appeared. He said that he had a patient at Frankston with a somewhat similar lesion and that he was contemplating the advisability of sequestrectomy. His advice, however, was similar to that of Dr. Forster.

DR. REGINALD WEBSTER said that the appropriate gland for biopsy examination would be one of the external iliac group and not an inguinal gland. The proof of a tuberculous aetiology in an inguinal gland had a bearing on the aetiology of conditions in the knee joint, but not on those of the hip joint.

DR. K. H. HALLAM said that, on inspecting the films shown by Dr. Stephens, his first impression was that the appearances were those of a tuberculous lesion; but on analysis he had observed three features that would in that case be unusual. They were: (i) that the disease was limited by the cartilage between the head and the neck of the femur, as in coccal lesions; (ii) that there was expansion in the neck, which was not a feature of tuberculous infection; (iii) that there was no evidence of synovial involvement. He concluded, however, that they must be influenced by the personal and family history, and must be persuaded that the lesion was tuberculous.

DR. Stephens, in reply, said that he had felt that the lesion was tuberculous since December, 1940, but the skiagraphic examination had opened up the other problems; it was unusual, for example, to find what he took to be the appearances of sequestration. The child had worn knock-knee splints for nine months, and Dr. Stephens thought it possible that the trochanteric portion of the knock-knee splint had created irritation. He could recall the occurrence of osteomyelitis near the knee joint following the treatment of knock-knee and appearing at the site at which pressure from the straps was applied. In the case of the child under discussion the lesion was extraarticular; but it was impinging on the epiphyseal cartilage, and if the sequestrum was attacked, the blood supply to the head and neck of the bone might be diminished. Dr. Stephens would have to remove an iliac gland for investigation; but he had not done so, as he had expected to make the opening over the trochanter. If the lesion was a little further from the cartilage, he would still be inclined to operate; but as things were, he proposed to adopt conservative measures. He added that numerous leucocyte counts had been made, but had carried no diagnostic significance.

#### Drawing Tack in the Lung.

DR. J. G. WHITAKER showed a boy, aged nine years, who had had a drawing tack in the lung. The occurrence of several febrile attacks had led to radiographic investigation of the chest, and the presence of the tack had been established by examination of the films. Dr. Raymond Hennessy had on several occasions attempted to extract it, but could not reach it, and the boy had come into the hands of Dr. Whitaker in the expectation that the tack was accessible surgically. The boy had been under treat-

ment for two and a half years. Dr. Whitaker had operated in December, 1940, and removed the tack. The condition of the patient before operation was quite good. Dr. Colin Macdonald had also been interested in the patient and had insisted that the continued presence of a metallic foreign body in that position in the lung was not of much importance. Dr. Howard Williams had carried out the preliminary induction of pneumothorax, and though the boy had had rather heavy dosage of morphine and hyoscine immediately before the operation, he was not in a deeply depressed state. An intercostal incision was made; this allowed adequate exposure of the pleura, and it was necessary only to cut a little way into the lung, remove the tack and sew up the incision. The chest was closed under a negative pressure system and convalescence had proved uneventful.

DR. WHITAKER observed that, as the tack was in a subpleural position, it was apparent at the operation that it could not have been removed through a bronchoscope by DR. HENNESSY. He had also been impressed by the absence of any reactionary lesion in the surrounding lung tissue, and the boy had had very little ill-health and no expectoration of sputum. Since the operation he had been quite well and the lung had expanded readily.

DR. H. L. STOKES, from the chair, congratulated DR. Whitaker on the very happy result in a perplexing case.

DR. GUY SPRINGTHORPE asked for information concerning the probable happenings if such a foreign body was left in situ.

DR. W. KENT HUGHES said that it was difficult to understand why it had not been thought sufficient to disperse the granulations by the electro-cautery. He added that he had been amused, when going through the Panama Canal in 1940, to find that Australia seemed to be known there only as the place from which a patient with a foreign body in the lung had to be sent to the United States of America to have it removed.

DR. K. HALLAM reminded the members that at a previous meeting he had urged strongly that the tack should be left alone if there was no evidence of damage in the lung. If a foreign body was in the lung and was not causing an inflammatory reaction, it should be left alone. It was of interest to him to hear from Dr. Whitaker that there was no evidence of inflammatory reaction. He thought that the child would still be very well if there had been no operation.

DR. WHITAKER, in reply, said that he could not answer DR. Springthorpe's question, though the point had appealed to him. The patient had for two years prior to the operation had frequent pneumonic attacks; but during and after the operation Dr. Whitaker had been struck by the fact that he was not doing much good by removing the tack. He added that the necessity to send the patient to America had happily been averted.

#### Partial Removal of Horseshoe Kidney.

DR. WHITAKER also showed a girl, aged three years, who had undergone partial nephrectomy. He said that the case illustrated the great importance of the thorough urogenital investigation of patients suffering from pyuria. The patient had been referred to him after having been in the care of DR. D. M. Emberton and DR. J. W. Grieve. At the preliminary cystoscopic investigation Dr. Whitaker had obtained pus and blood from each ureter and had performed retrograde pyelography. He showed the unusual appearances in the pyelograms; these had suggested that a horseshoe kidney was present and that the left side of it or the left kidney was the abnormal portion. He went on to say that at the operation he had used the transverse incision and the intraperitoneal route, approaching the kidney through the peritoneum from the front. He had tied off the vessels before he was able to be certain that the kidneys were fused, and had removed the left portion of a horseshoe kidney after ligation near the point of fusion. Three months had elapsed since the operation and no further feverishness had occurred. It was of interest to attempt to forecast the subsequent course of the condition, and he wished to stress the value of the transverse incision and the approach, which rendered it easy to deal with the kidney satisfactorily.

DR. E. E. PRICE agreed with DR. Whitaker's advocacy of the transverse incision. He said that it was a great advantage to the surgeon to be able to see so well what he was about to do.

DR. W. R. FORSTER raised a doubt as to whether the kidney was really of the horseshoe type. After studying the pyelograms, he had formed the opinion that the left kidney might be a double one, like a bilobed kidney, and that one of the lobes was the infected one; there appeared to be two separate sections in the left kidney markings, and there

might easily have been two ureters. He added that the irregularity of the pelvis might have predisposed to the production of the infection.

Dr. Stephens said that the mother had told him that the child was still passing turbid urine containing pus, and asked what further treatment was to be carried out. He wondered whether the child was doomed to continued infection and ultimately to uremia.

Dr. Whitaker, in reply, said that the approach to the treatment had been quite simple; the child had gross pyuria and numerous severe febrile attacks, and he had been able to obtain pyelographic demonstration of abnormality on the left side. These were indications to operate on the left side. He went on to say that there was a calyx crossing the mid-line at the site of ligation, but no leak had occurred. He had had previous experience of bilateral double ureter, but no double ureter was present in the case under consideration. Dr. Whitaker also mentioned that permission for operation had been refused by the parents of the patient who had a double ureter, because the attacks of illness were so mild. Dr. Whitaker, in conclusion, said that any further treatment would have to be medical treatment.

#### Tuberculous Disease of the Spine.

DR. REGINALD WEBSTER showed a specimen of tuberculous disease of the spine, in order that he might take up its story at the point where he had left it at a former meeting. The specimen was secured by Dr. Stella Altmann at the autopsy on a girl, aged four years, who had died at the orthopaedic section of the hospital at Frankston.

In May, 1939, this girl had been admitted to the Children's Hospital, presenting an indolent submental ulcer, which subsequent investigations on a neighbouring lymphatic gland had shown to be tuberculous in origin. Cultures of *Mycobacterium tuberculosis* recovered from the gland were characteristically bovine in type. There was nothing unusual in such a finding, as Dr. Webster's experience had shown the tonsils and cervical glands to be the happy hunting ground of bovine tubercle bacilli in their activities among children.

The child was duly discharged from the hospital, but returned in July, 1939, with clinical and radiological evidence of tuberculous disease of the spine. She was transferred to Frankston, where she died in August, 1940. From the destructive lesion of the first lumbar vertebra which was to be seen in the specimen, he had cultivated *Mycobacterium tuberculosis*; it had also been grown from a specimen of cerebro-spinal fluid which Dr. Altmann had collected.

On the occasion of the former meeting Dr. Webster had not determined the type of *Mycobacterium tuberculosis* present in the spine and cerebro-spinal fluid, but he had ventured to predict that it would be the same as that recovered from the cervical gland. He based this anticipation on a statement of A. S. Griffith, who, writing in 1928, said that among 1,405 cases in which tubercle bacilli had been recovered from different sources in the same individual, there had been no instance of mixed infection.

The cultures of *Mycobacterium tuberculosis* recovered from the spine and cerebro-spinal fluid, however, were just as indubitably of human type as that from the gland of the neck was bovine. In his opinion there was no possibility of error, the contrast in the cultures from the cervical gland and the spine respectively being very obvious. The growths from the cervical gland were typically bovine in their dysgonic character, hesitant and receding in artificial culture, but aggressive and virulent when introduced into the circulation of a rabbit. Cultures from the spine and cerebro-spinal fluid were conversely luxuriant and flamboyant in artificial culture and of comparatively low virulence in the rabbit. The bovine strain killed a rabbit in nineteen days by inducing severe and widespread tuberculosis; a rabbit inoculated with the human strain lived for ninety days, and when it was killed sparsely distributed and retrogressive lesions were found. It was now to be shown that this little girl had made a posthumous contribution to bacteriological history.

A. Stanley Griffith, in 1937, in a general review of the subject of mixed infection with human and bovine types, emphasized that it was rare to find more than one type of tubercle bacillus in a tuberculous human subject. Observations in Germany, Denmark and Great Britain, the three countries in which the most extensive studies of the types of tubercle bacilli occurring in human disease had been made, furnished the basis for this statement. In Germany, Lange had found that both types were present in only eight of 1,027 cases bacteriologically examined up to the year 1932. K. A. Jensen, in Denmark, had met with only six instances of mixed infection among 3,000 examples of tuberculosis in human subjects. In all six observed by

Jensen the patients were affected with pulmonary tuberculosis, and both human and bovine types of tubercle bacilli were present in the sputum. In Great Britain mixed infections with human and bovine bacilli had been recorded in 10 among 6,000 tuberculous persons concerning whom the necessary examinations had been made. In all except one the two types had been associated in the same organ or gland. The one exception had been reported by Blacklock in his well-known monograph on tuberculosis in childhood; a boy who died from tuberculous meningitis provided a human strain from a tracheo-bronchial gland and a bovine strain from a mesenteric gland.

Griffith proceeded to report the details of an example of mixed infection which had come under his own notice. The patient was a young woman, aged eighteen years, who had died from tuberculous meningitis. The bovine type was found present alone in the meninges, and both human and bovine bacilli were recovered from the lung. This was a second example of differing types of bacilli recovered from tissues at a distance from each other, and the child from whom Dr. Webster had obtained the specimen under consideration furnished a third. She had yielded the bovine type from a gland in the neck and the human type from the diseased vertebra and the cerebro-spinal fluid. Thus very few genuine examples of mixed infection were on record, and it appeared that the chance of its occurring was about 1 in 500, always provided that cultures were made from multiple sources in autopsy work on tuberculous patients. In numerous cases, however, cultures had been obtained from lesions in more than one organ or gland, and except in those to which Dr. Webster had referred, the multiple strains from an individual patient had always been identical in cultural characters and virulence. Coexistence of viable human and bovine strains in human tuberculosis was therefore very rare.

#### Pes Cavus.

DR. E. E. PRICE showed a series of children to demonstrate the applicability of what he called "Lambrinudi's rather revolutionary procedure" for the improvement of patients suffering from *pes cavus*. Dr. Price first contrasted the two main types of the deformity. He said that in the calcaneo-cavus type, the back foot dropped from paralysis of calf muscles and adaptive shortening of the structures in the sole of the foot resulted in its being hollowed. On the other hand, in equino-cavus it was the forefoot which dropped. That condition seemed to be the end-result of different causes, such as paralysis, spasticity and certain unclassified or idiopathic factors. When the metatarsal heads presented to the ground, a varying degree of clawing of the toes aggravated the metatarsal prominence. Dr. Price added that the mode of production of that deformity was still debatable; for the purpose of demonstration, however, he wished to point out two facts. If the toes of a healthy foot were retracted, the arch was increased; and in equino-cavus, if the metatarsal heads were pushed up, the toe retraction as well as the cavus condition could be reduced or in early cases abolished. It was difficult to maintain the straight position of the toes; but efforts in that direction would also reduce the cavus. Thus it was possible to postulate that retraction of the toes was the primary step in the production of the deformity.

Dr. Price went on to say that that postulate appeared to be the theoretical basis for Lambrinudi's operation, a description of which could be found in "Recent Advances in Orthopaedic Surgery", by Burns and Ellis, 1937, at pages 273 to 276. The principles of the operation were to prevent clawing by arthrodesis of the interphalangeal joints with tenotomy of the extensor tendons and fixation of the toes to a metal splint by means of skeletal traction, in which sutures of silkworm gut were passed round the dorsum of each proximal phalanx.

From the aspect of treatment it was convenient to classify the patients into three groups. In early cases the deformity could be corrected if the metatarsal heads were pushed up; a night-shoe for cavus and a metatarsal bar were used. Next came the moderate cases, in which there was some fixed deformity; and that group of patients could be subdivided into those with definite toe deformity, for whom the Lambrinudi procedure was suitable, and those with pronounced plantar contracture associated perhaps with less pronounced clawing, for whom the Lambrinudi procedure was not suitable and for whom the Steindler procedure would be appropriate. The third group of patients comprised those with advanced bony deformity. They required stabilization operations. The varus deformity was usually a feature to be taken into account.

Dr. Price then showed a patient upon whom he had performed an operation which was not exactly that described in the article he had quoted. The steps of his operation

were the following. A dorsal longitudinal incision was made on each toe, through which the dorsal extensor tendon was divided opposite the metatarso-phalangeal joint and dissected distally, being afterwards retained as a retractor. He had proceeded to capsulotomy of the metatarso-phalangeal joints to a degree sufficient to correct hyperextension; that procedure might involve the lateral ligaments. He said that the hallux received special attention; the tendon of the *extensor hallucis longus* muscle was grafted to the neck of the first metatarsal bone towards the end of the operation. After capsulotomy, Dr. Price said he arthrodesed each of the interphalangeal joints, though if the distal phalanges were small it was often better to leave the distal joints alone. He added that the little toe might be treated by arthrodesis or by removal of a phalanx, or it might be left untreated. The next step was that of fixation of the toes to the splint. He said that sutures of silkworm gut or of strong silk were threaded on a straight needle and passed close to the proximal phalanx and out through the plantar aspect of the toe; the other end was threaded and used similarly, so that a loop was thrown across the phalanx. At that stage the incisions were closed by suture. Then a sterilized aluminium plate of suitable size, cut with five prongs for the five digits and padded with sterile felt or sponge rubber over the region of the tread, was placed in position and fixed by means of a plaster cast which corrected the cavus and maintained slight pressure on the corrected foot. Finally, the long sutures were tied over sterile pads, each toe being anchored to a separate prong of the splint.

Dr. Price explained that the operation was performed with a tourniquet in position, and each foot took between forty-five and sixty minutes; one foot was quite enough at a time, though both could be attended to at one sitting if necessary. He explained that the plaster cast was removed within eight weeks; the first steps was the cutting of the sutures, which could be withdrawn when the cast was off the foot.

Dr. Price showed a series of patients, including one each of whose feet had been treated in 1940, a twelve year old girl undergoing treatment who had the foot-plate and cast in position, and a girl upon whom he proposed to operate.

DR. KENT HUGHES said that Lambrinudi's operation did not relieve cavus, but was merely an aesthetic procedure for clawed toes and did not in any way improve efficiency. He explained that clawed toes did not seriously impair the function of the foot, except in so far as they produced intractable corns, and the clawing often disappeared when the cavus was abolished. He advanced the further objection that it seemed to him unnecessary to destroy the joints of the phalanges and interfere with the musculature; the patient was deprived of the natural functions of the muscles of the toes without gaining the slightest reduction of the cavus.

Dr. Kent Hughes said that a much simpler operation for clawed toes was one in which all the dorsal and plantar flexor tendons of the toes were divided subcutaneously and the contracted capsules were broken down or divided if necessary. The long dorsal flexor tendon of the first digit might take weeks to function, but non-union had never occurred in his experience. The patient shown by Dr. Price with cavus following upon anterior poliomyelitis had *pes cavus*, and dorsal flexion was limited to 90°. If the cavus had been dealt with it was probable that the clawing would have disappeared; Dr. Kent Hughes was afraid that the patient would have serious trouble later on. In the case of the other patient with inherited clawed foot, there was dorsal flexion of 90°, but no cavus, and he doubted whether an operation was necessary. The other patient who was to have an operation had only a suspicion of clawing, and if Dr. Price would consider systematic stretching of the calcaneous tendon by means of exercises, no further treatment would be necessary. Dr. Kent Hughes then said that diminution of dorsal flexion was very common in the young adult males he examined for the Tramways Board, and he advised against employment of any with less than 80° of dorsal flexion; consequently flat feet, sprained ankles and strained knees were rapidly disappearing from the service. He remarked that diminution of dorsal flexion was in his opinion the most frequent of hereditary defects. It seldom appeared before the subject was seven years of age, and frequently did not develop into a disability until the age of twenty years, though there was no relation between the degree of diminution and the amount of cavus, and gross cavus might occur without shortening of the calcaneous tendon. Dr. Kent Hughes added that, sixty years earlier, Redard had suggested that cavus was due to an inherent shortening of the intrinsic muscles of the foot; as there was an absence of dorsal flexion in all hooved animals, it was probable that shortening of the calcaneous tendon and of the plantar muscles was due to atavism. He

went on to say that the hamstring tendons were another set which was frequently shortened, whether from walking with bent knees or because of inherent defect it was hard to decide. In conclusion, Dr. Kent Hughes referred those interested in *pes cavus* and its treatment to an article he had written, which was published in *The British Medical Journal* of December 28, 1940.

DR. J. G. WHITAKER congratulated Dr. Price on an excellent and beautiful result in the case of the patient who had been treated in 1940. He also thanked Dr. Kent Hughes for his instructive remarks. He added that Dr. Price had combined correction of *pes cavus* with the undoing of the claw condition.

DR. H. DOUGLAS STEPHENS asked Dr. Price to arrange to show the same children again about a year later and to present a review of the subject then. He then referred briefly to idiopathic *pes cavus*, mentioning a girl, aged thirteen years, in his care at the Children's Hospital, who had had no *pes cavus* till she was aged eleven years; but he had found a mole present at the usual site of *spina bifida occulta*. Dr. Stephens also referred to a boy of about the same age who had developed *pes cavus* but had no clinical evidence of *spina bifida occulta*; from the skiagrams, however, it could be seen that there was non-union of the spines of the two lowest vertebrae. He had formed the opinion that the spinal cord became pulled up with tension of the fibrous band, and had wondered whether some lesion of that kind was responsible for the production of *pes cavus* in certain otherwise obscure cases.

DR. STOKES, from the chair, thanked Dr. Price for his demonstration and Dr. Reginald Webster for still another of his excellent pathological demonstrations.

Dr. Price, in reply, said that he was afraid that the subject of *pes cavus* was as callous as the soles of the feet of the patients. He had found the remarks of Dr. Kent Hughes very instructive and he expressed his gratitude for them. He explained that the patient he had treated and the one he was treating were the only children he had undertaken to treat up to the time of the meeting, so that the cases must be regarded as essentially experimental. He had gained his knowledge and experience chiefly in connexion with an adult clinic at the Royal Melbourne Hospital. The condition in adults was usually associated with foot strain, and he had translated the technique adopted at the adult hospital to suit the needs of children. His object was to undertake treatment at a much earlier stage in the development of the condition, in the expectation that good results could be obtained and a lot of unnecessary suffering eliminated. He said that he would be glad to review the position in a year or so, and would try to show the same patients again on that occasion.

## Correspondence.

### EAR PICKING AND EYE CLEANING IN THE MIDDLE AND FAR EAST.

SIR: I was very interested in the article in *THE MEDICAL JOURNAL OF AUSTRALIA*, April 5, on "Ear Picking and Eye Cleaning in the Middle and Far East", by Isadore Brodsky. The description of the instruments and Mukhopadhyaya's statement that the barbers of India now use an ear scoop for extracting wax reminded me that I once saw a pair of these ear scoops.

On April 21, 1938, I was consulted by an Australian-born Chinese from Innisfail, re his ear condition. After giving him the age-worn advice "not to put anything smaller than his elbow in his ear" and also going through the list of things people usually use in an ear, such as pins, pencils, pens, paper clips, tooth-picks, crochet needles, matches, bobble clips *et cetera*, I was rather amazed to be told in perfect English that he used the correct instrument for extracting wax and nothing else.

On inquiry, I found he used ear scoops, which he brought down for me to see on his next visit.

From memory, these were two silver scoops about three (3) inches long. The handle was modelled or carved and the blade or scoop itself somewhat flattened, like a bone mustard spoon. The handle carried a little ring, by which it could be attached (I wonder if you would wear it on your watch-chain or key-ring).

However, I felt it my duty to warn him that the use of even such an instrument was unwise in this land of intense humidity and heat, owing to the prevalence of otomycotic conditions and allied furunculosis.

Yours, etc.,

C. E. S. JACKSON.

Townsville,  
Queensland,  
April 8, 1941.

*Post scriptum.*—I wonder if Charles Chaplin knew that Eastern barbers removed wax. I refer to the scene in the barber's shop in the "Great Dictator", when, after shaving the customer, he looked in his ear and put his finger in the customer's ear as if trying to remove wax.—C.E.S.J.

#### THE TOURNIQUET AND BOTH BONES.

SIR: It appears that many doctors as well as first-aid men still teach that an effective tourniquet cannot be applied round both bones of the leg or forearm. This is but a perpetuation of text-book doctrine without testing its truth, for it can easily be disproven in the case of an elastic tourniquet. A beautiful white limb can be obtained, after a few minutes' elevation, by its application round the forearm or leg; indeed, I fancy it is rather easier to apply effectively to the calf than to the thigh, and am prepared to demonstrate it to anyone who cannot convince himself.

Yours, etc.,

CRAWFORD MCKELLAR.

Harley,  
143, Macquarie Street,  
Sydney.  
April 12, 1941.

#### THE ILLAWARRA SUBURBS MEDICAL PRACTITIONERS' NATIONAL EMERGENCY SERVICE.

SIR: The enclosed statement of receipts and expenditure of the Illawarra Suburbs Medical Practitioners' National Emergency Service for the year ended April 8, 1941, may be of some interest to the members of the profession.

The scheme was evolved shortly after the declaration of war, and was in readiness to operate immediately upon the enlistment of any of our members in the permanent forces.

The scheme provides that the lodge lists of practitioners on active service shall remain unaltered for the duration of the war, the remaining members undertaking the care of these patients, whilst the capitation fees are paid direct to the representatives of the absentee practitioners by the lodges concerned. In addition, each remaining practitioner has undertaken to pay five shillings per week to every member who is away on active service. This money is paid quarterly by bank authorities to facilitate collection.

The liability of the remaining practitioners is limited to the sum of three pounds per week.

#### ILLAWARRA SUBURBS MEDICAL PRACTITIONERS' NATIONAL EMERGENCY FUND. Statement of Receipts and Expenditure for First Year ended April 8, 1941.

RECEIPTS.	f	s.	d.
Contributions by 44 members	2,073	12	6
Bank Interest to June 30, 1940	0	13	2

£2,074 5 8

At the present time six members are receiving benefit under the scheme, each remaining member contributing £1 10s. weekly to the fund.

The first member called up was a member of the Australian Naval Reserve, and was given very little time to vacate his practice. The scheme, however, worked very smoothly and has continued to work very successfully.

Yours, etc.,

G. W. ASHBY,

Honorary Secretary,  
Illawarra Suburbs Medical Association.

80, Penshurst Street,  
Penshurst,  
New South Wales.  
April 17, 1941.

#### Naval, Military and Air Force.

##### APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been notified in the *Commonwealth of Australia Gazette*, Number 77, of April 17, 1941.

##### ROYAL AUSTRALIAN AIR FORCE.

###### Citizen Air Force: Medical Branch.

Flight Lieutenant W. D. Cunningham is transferred from the Reserve to the Active List, with effect from 17th March, 1941.

###### Reserve: Medical Branch.

The appointment of Flight Lieutenant J. A. Bassetti is terminated, with effect from 1st October, 1940.

The following are granted commissions on probation, with the rank of Flight Lieutenant, with effect from the dates indicated: John Dean Bishop, M.B., B.S., 28th March, 1941; Neil Oldfield, M.B., B.S., and Frank Haughton Lord, M.B., B.S., 21st March, 1941.—(Ex. Min. No. 54—Approved 16th April, 1941.)

#### Australian Medical Board Proceedings.

##### NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act, 1938-1939*, of New South Wales, as duly qualified medical practitioners:

McCallum, Norman Yates, M.B., B.S., 1940 (Univ. Sydney), General Hospital, Brisbane.  
Morelini, John Allan, M.B., B.S., 1940 (Univ. Melbourne), c/o Dr. Pfeiffer, 29, Anglo Road, Campsie.

EXPENDITURE.	f	s.	d.
Distribution—			
Dr. "A", enlisted April 8, 1940 .. ..	499	13	11
Dr. "B", enlisted May 8, 1940 .. ..	424	2	2
Dr. "C", enlisted May 20, 1940 .. ..	392	16	8
Dr. "D", enlisted June 27, 1940 (subsequently obtained locum) .. ..	36	18	2
Dr. "E", enlisted October 4, 1940 .. ..	236	2	6
Dr. "F", enlisted January 28, 1941 .. ..	55	2	6
Dr. "G", enlisted February 15, 1941 .. ..	21	7	6
	1,666	3	5
Expenses—			
Accountancy, notifying Lodge Secretaries, Stamp Duty on Bank Authorities, and General Clerical Expenses in operating Scheme, including Postage and Stationery			
Balance in Hand (due to Subscriptions collected quarterly in advance)	95	0	5
	313	1	10
	£2,074	5	8

Miller, Elizabeth Constance Graham, M.B., B.S., 1937 (Univ. Melbourne), L.M.S.S.A., 1939, 21, Market Street, Goulburn.

Allardyce, Ransome Macnamara, M.B., B.Ch., 1926 (Univ. Dublin), 6, Union Street, Mosman.

Thomson, John Douglas, M.B., Ch.M., 1937 (Univ. New Zealand), St. James' Flats, Stanley Street, Sydney.

The following additional qualifications have been registered:

Puckey, Mary Courtenay, Sydney (M.B., Ch.M., 1923, Univ. Sydney), D.P.H., Univ. Sydney, 1925.

Main, James Norman, Orange (M.B., Ch.M., 1924, Univ. Sydney), D.P.M., Univ. Sydney, 1940.

The following change of name has been registered:

Rippin, Muriel, Adaminaby, now McPhillips, Muriel.

## Obituary.

### JOHN FERGUSON CHAMBERS.

We regret to announce the death on active service of Major John Ferguson Chambers, of the Australian Army Medical Corps.

### ZELMAN SCHWARTZ.

We regret to announce the death on active service of Major Zelman Schwartz, of the Australian Army Medical Corps.

## Medical Appointments.

Dr. John Robert Thompson has been appointed quarantine officer at Port Augusta, South Australia, under the provisions of the *Quarantine Act, 1908-1924*.

Sir Raphael West Climento has been appointed a Member of the Senate of the University of Queensland, in terms of section 12 of *The University of Queensland Act of 1909*.

Dr. Walter Ernest Summons and Dr. Sidney Valentine Sewell have been appointed Members of the Commission of Public Health, under the provisions of the *Health Act, 1928*, of Victoria.

Dr. Frank Kenneth Mugford has been appointed Medical Officer to the Children's Welfare and Public Relief Department, under the provisions of the *Public Service Act, 1936-1940*, of South Australia; Medical Officer, Yatala Labour Prison and the Adelaide Gaol, under the provisions of the *Prisons Act, 1936*, of South Australia; Medical Officer, Yatala Labour Prison and the Adelaide Gaol, under the provisions of the *Criminal Law Consolidation Act, 1935-1940*, of South Australia.

Professor John Burton Cleland has been appointed a Member of the Fauna and Flora Board of South Australia.

Dr. Alexander Jamieson Meikle has been appointed an Official Visitor to the Parkside Mental Hospital, South Australia.

Dr. John Robert Thompson has been appointed Medical Officer to the Port Augusta Hospital, South Australia.

Dr. Francis Patrick Quirk has been appointed Government Medical Officer at Peak Hill, New South Wales.

Sir Norman Paul has been appointed a Member and Vice-Chairman of the Hospitals Commission of New South Wales, pursuant to the provisions of the *Public Hospitals Act, 1929-1940*, of New South Wales.

## Books Received.

"Illustrations of Regional Anatomy", by E. B. Jamieson, M.D.; published in five sections; Third Edition; 1941. Edinburgh: E. and S. Livingstone. Royal 8vo. Price 36s. 6d. net.

"Surgery of Modern Warfare", edited by H. Bailey, F.R.C.S.; Part III; 1941. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 159, with illustrations. Price: 12s. 6d. net.

## Diary for the Month.

MAY 6.—Queensland Branch, B.M.A.: Post-Graduate Committee.  
 MAY 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.  
 MAY 7.—Western Australian Branch, B.M.A.: Council.  
 MAY 7.—Victorian Branch, B.M.A.: Branch.  
 MAY 9.—Queensland Branch, B.M.A.: Council.  
 MAY 9.—Victorian Branch, B.M.A.: Legislation Subcommittee.  
 MAY 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
 MAY 13.—Tasmanian Branch, B.M.A.: Branch.  
 MAY 15.—Queensland Branch, B.M.A.: Ipswich Hospital Clinical Society.  
 MAY 15.—Queensland Branch, B.M.A.: Hospital for Sick Children Clinical Society.  
 MAY 15.—Victorian Branch, B.M.A.: Ethics Subcommittee.  
 MAY 19.—Victorian Branch, B.M.A.: Hospital Subcommittee.  
 MAY 20.—New South Wales Branch, B.M.A.: Ethics Committee.  
 MAY 20.—Victorian Branch, B.M.A.: Organization Subcommittee.  
 MAY 20.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.  
 MAY 21.—Western Australian Branch, B.M.A.: Branch.  
 MAY 22.—New South Wales Branch, B.M.A.: Clinical.  
 MAY 22.—Victorian Branch, B.M.A.: Executive.  
 MAY 23.—Queensland Branch, B.M.A.: Council.  
 MAY 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phoenix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and book-sellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £3 for Australia and £1 5s. abroad per annum payable in advance.